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CHARACTERISATION OF ELECTROMYOGRAPHIC ACTIVITY IN NORMAL AND PATHOLOGICAL KNEE JOINTS DURING SEATED EXTENSION

by

RAY MARKS

A THESIS

SUBMITTED TO THE FACULTY OF GRADUATE STUDIES AND RESEARCH IN PARTIAL FULFILMENT OF THE REQUIREMENTS FOR THE DEGREE

of

Master of Science

Department of Physical Therapy

EDMONTON, ALBERTA,

SPRING 1988

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The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled "CHARACTERISATION OF ELECTROMYOGRAPHIC ACTIVITY OF THE QUADRICEPS MUSCLE IN NORMAL AND PATHOLOGICAL KNEE JOINTS DURING SEATED EXTENSION" submitted by Ray Marks in partial fulfilment of the requirements for the degree of Master of Science in Physical Therapy.

(Supervisor)

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Date: April 27, 88

DEDICATION

To my parents and Phil for his unceasing loyalty and support

ABSTRACT

Osteoarthritis (OA) is a progressive and disabling disorder characterized by pain, stiffness, instability, deformity, limitation of motion and loss of function in one or more joints. The role of muscle in the etiology of the disease is unclear, although muscle weakness, biopsy and electromyographic changes are found to occur in association with affected joints.

The objective of this study was to compare the electromyographic (EMG) activity from selected points of the anterior knee joint musculature, amongst subjects with normal knee joints and those subjects presenting with osteoarthritis of the knee joint. In addition, normal healthy subjects presenting with tibial malalignment were evaluated as a separate "risk" group.

Subjects in all groups attended a single test session during which full wave rectified and linear envelope detected EMG recordings were made with respect to the vastus medialis (VM), vastus lateralis (VL) and rectus femoris muscles (RF) during trials of isometric and isokinetic knee extension.

Results of peak and integrated EMG (IEMG) recordings indicated a number of significant differences between groups and muscles studied. In particular, normalized EMG activity was higher in arthritic subjects during isometric

and isokinetic contraction and higher IEMG/torque ratios during isometric contraction were recorded. In addition, time factors related to tension development were significantly reduced in this group with respect to both isometric and isokinetic contractions.

The RF muscle showed significant differences in its activity patten with respect to peak EMG values recorded by arthrtic subjects and peak activity was increased during isometric and decreased during isokinetic contraction.

EMG values and time factors related to tension development showed similarities of activity between risk and arthrtic subjects in some instances, in others, the risk group was found to perform normally.

Results of this preliminary investigation suggest that the adaptive responses of the musculature in OA are complex. The potential for early prediction of OA has also been assessed. Further study is needed before clinical implications can be derived.

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Table of Contents

CHAPTER	PAGE
I. INTRODUCTION	1
A. BACKGROUND AND RATIONALE	1
B. EVALUATION OF MUSCLE FUNCTION IN OA	5
C. OBJECTIVES OF STUDY	6
D. RESEARCH HYPOTHESIS	7
E. SIGNIFICANCE OF STUDY	8
F. OPERATIONAL DEFINITIONS	9
G. DELIMITATIONS	12
H. LIMITATIONS	13
II. REVIEW OF THE LITERATURE	14
A. INTRODUCTION	14
B. OSTEOARTHRITIS	15
Classification	17
General Prevelance	18
Natural History	20
Mechanical Factors	23
Muscular Reflex Mechanisms	26
Muscle Pathology	27
Muscle Imbalance	30

C. THE KNEE JOINT AND OA 32
KNEE JOINT STRUCTURE AND FUNCTION 34
Bony Structure 35
Articular Cartilage: Histology 35
Articular Cartilage: Function 37
Articular Cartilage: Pathology 39
Knee Joint Musculature 40
Biomechanics 42
Knee Muscle Imbalance 44
D. EVALUATION OF OSTEOARTHRITIS 46
Pertinent studies 47
Strength Measurements 47
Electromyographic Studies 49
Gait Analysis 51
E. ELECTROMYOGRAPHY 52
Surface Electrodes 54
Placement 56
Skin Preparation 57
Amplification 58
Processing 59
Normalization 60
EMG and Muscle Tension 61
Control of muscular force 62
Length-tension relationship 63
Influence of velocity 63
Electromechanical delay 64

Activity of knee joint extensors 66
F. TESTING WITH AN ISOKINETIC DYNAMOMETER ... 68
Reliability and validity of the Cybex 69
Measures from the torque curve 69
The strength torque curve 72
Force velocity curves 73
G. PHYSIOTHERAPY IN OA 73
III. METHODS 78
A. SUBJECTS 78
Inclusion Criteria 78
Exclusion Criteria 79
B. RESEARCH DESIGN 79
C. EVALUATIVE PROTOCOL 81
Clinical 81
Function 83
EMG Measures 83
Torque Measures 83
D. TESTING 83
E. MEASURES..... 98
F. DATA ANALYSIS 98
G. ETHICAL CONSIDERATIONS 100
IV. RESULTS AND DISCUSSION 102
A. Subjects 102
B. Electromyographic data 107
a) Peak EMG 107
b) Raw EMG 119

c) IEMG data 129
d).IEMG/force data 138
C. Time Factors 146
a) Rate of isometric tension dev. 145
b) Rate of isokinetic tension dev. 151
c) Initial rate of tension dev. 153
d) Electromechanical delay 156
e) Time to peak Isokinetic EMG 158
D. Torque measures 162
a) Normalized torque values 162
b) Initial tension development 166
Summary 169
CLINICAL IMPLICATIONS 173
V. SUMMARY AND CONCLUSIONS..... 176
Recommendations..... 181
REFERENCES 182
APPENDIX A: CLASSIFICATION OF OA..... 206
APPENDIX B: RADIOGRAPHIC CLASSIFICATION..... 209
APPENDIX C: FUNCTIONAL QUESTIONAIRRE..... 211
APPENDIX D: KNEE ASSESSMENT FORM..... 214
APPENDIX E: SUBJECT CONSENT FORM..... 217
APPENDIX F. CYBEX CALIBRATION..... 220
APPENDIX G: AMPLIFIER SPECICATIONS..... 223
APPENDIX H: CONFIRMATION LETTER..... 225
APPENDIX I: AMPLIFIER CALIBRATION..... 227
APPENDIX J: RAW DATA..... 230

APPENDIX K: ANOVA SUMMARY TABLES.....242

APPENDIX L: RELIABIILTY COEFFICIENTS.....250

LIST OF TABLES

Table		Page
2.1	Thigh muscle weakness in pathology.....	48
4.1	Anthropometric Data.....	104
4.2	Peak isometric EMG at 30 degrees.....	108
4.3	Peak isokinetic EMG	109
4.4	Isometric IEMG at 30 degrees	131
4.5	Isokinetic IEMG	133
4.6	Isometric IEMG/force ratio at 30 degrees	140
4.7	Isokinetic IEMG/force ratio.....	141
4.8	Isometric TRTD.....	148
4.9	TRTD isokinetic.....	152
4.10	Initial rise of torque curve.....	155
4.11	Isokinetic EMD.....	156
4.12	Isokinetic TTP EMG.....	159
4.13	Isometric normalised torque.....	164
4.14	Isokinetic normalised torque values.....	166
4.15	Initial Isometric torque	168

LIST OF FIGURES

Figures		Page
2.1	Knee extension curve.....	71
3.1	Sample trace isometric.....	97
3.2	Sample trace isokinetic.....	99
4.1	Comparison of isometric peak EMG.....	110
4.2	Comparison of isokinetic paek EMG.....	112
4.3	Comparison of isometric IEMG.....	130
4.4	Comparison of isokinetic IEMG.....	132
4.5	Comparison of IEMG QF.....	134
4.6	Comparison of isometric IEMG/force.....	139
4.7	Comparison of isokinetic IEMG/force.....	142
4.8	Comparison-isometric;isokinetic IEMG/force	144
4.9	Comparison of TRTD isometric.....	147
4.10	Comparison of TRTD isokinetic.....	150
4.11	Comparison of Initial isometric TRTD....	154
4.12	Comparison of isokinetic EMD time.....	157
4.13	Comparison of isokinetic TTP EMG.....	160
4.14	Comparison of normalised isometric torque	162
4.15	Comparison of normalised isokinetic torque	165
4.16	Comparison of initial isometric torque .	167

LIST OF PLATES

Plate		Page
3.1	Emg amplifier.....	84
3.2	Isokinetic dynamometor.....	84
3.3	Subject positioned for isokinetic contration	88
3.4	Subject postioned for isometric contraction	88
3.5	Knee positioned at 60 degrees.....	90
3.6	Subject postioned for isometric contraction	91
3.7	Positioned for isometric contraction....	93
3.8	Stabilised for isometric contraction....	94
3.9	Axis of dynamometor aligned with knee axis	96
4.1	EMG and Torque record-normal subject, 60 deg.	120
4.2	EMG and Torque record-risk subject, 60 deg.	121
4.3	EMG and Torque record-OA subject,60 deg.	122
4.4	EMG and Torque record-normal subject,30 deg.	123
4.5	EMG adn Torque record-risk subject,30 deg.	124
4.6	EMG and Torque record-OA subject, 30 deg.	125
4.7	EMG and Torque record-normal subject,90deg/s	126
4.8	EMG and Torque record-risk subject,90deg/s	127
4.9	EMG and Torque record-OA,90 deg/s.....	128

LIST OF SYMBOLS AND ABBREVIATIONS

n	number
r	correlation coefficient
mm	millimetre
cm	centimetre
mv	millivolt
sec	second
deg/s	degrees per second
deg.	degrees
flex.	flexion
VM	vastus medialis
VL	vastus lateralis
RF	rectus femoris
QF	quadriceps femoris
EMG	electromyography
IEMG	integrated electromyography
IEMG/force	integrated electromyographic force ratio
PFS	patellofemoral syndrome
OA	osteoarthritis
RA	rheumatoid arthritis
ANOVA	analysis of variance
MVC	maximal voluntary contraction
TRTD	time rate of tension development
TTP	time to peak
lb.	pound

1. INTRODUCTION

A: BACKGROUND AND RATIONALE

Osteoarthritis (OA) is the most prevelant of the rheumatic joint diseases (1), occuring in one to two individuals of three in any population over 35 years of age (2). As defined by Doyle (3), OA is a degenerative condition affecting the axial skeleton and the peripheral diarthrodial joints in which there is progressive bone remodelling, outgrowth of bone, articular cartilage degeneration and capsular fibrosis. Symptoms of pain, joint stiffness, instability and swelling occur, resulting in functional disability (4). The increasing awareness that OA represents the largest proportion of the rheumatic joint diseases in a general population, coupled with the substantial economic and social cost (1) has resulted in a search for improved treatment strategies and a need to develop preventative measures against the disease. This has led to an intensified inquiry into the causes of this condition.

Although secondary osteoarthritic changes following trauma, congenital deformity, infection and other causes of joint incongruity are easily explained, there is as yet no clear explanation of the primary and more common form of this disease (4). A large body of research on possible chemical, enzymatic and hereditary factors has shown that

etiological relationship to primary degenerative joint disease. In addition, although primary joint disease is clearly age related (2), OA is not simply a process of aging (5).

From a clinical standpoint, primary OA is currently regarded by some as the manifestation of joint failure arising from a complex interplay of multiple factors (3,4,5). In particular, it is postulated that the disease may result from an imbalance between the mechanical stresses placed on the joint and the ability of the tissues of the joint to withstand these stresses (5,6). This may involve both static features of joint geometry, stability and alignment as well as the dynamic factors involving joint motion and sensory feedback mechanisms (5).

However, little imformation is available regarding the natural history of OA and the genesis of the disease is generally poorly understood, remaining largely conjectural despite significant research efforts in the past several years to increase our understanding of the condition.

One of the problems arising in studying the natural history of OA is the fact that the initiation of the disease is considered to be at a point quite remote in time from the clinical demonstration of the lesion and although the progress of the disease can be followed radiographically, thus monitoring characteristic features such as joint space loss, osteophyte formation, subchondral

sclerosis and cyst formation, these changes cannot be perceived until late in the disease. Therefore the in vivo characterisation and diagnosis of OA for either research or clinical purposes is difficult. Furthermore, the deficiencies in radiography present major difficulties in human clinical trials designed to evaluate the potential of therapy to alter the rate of progress of OA (7). Additionally, although bone scintiscanning and magnetic resonance imaging techniques show changes in OA earlier than those seen radiographically (7,8,9), these techniques are not universally employed for diagnostic purposes.

Since OA remains largely a clinical diagnosis for which back up evidence is provided by radiological investigation, and since joints are symptom free during the initial morphological changes of OA, patients with early signs of the disease may go unobserved and untreated as a result. Furthermore, a relatively weak relationship between radiographic findings and the symptoms of OA in weight bearing joints is found and forty percent of persons with radiographic evidence of OA may have neither disability nor symptoms of OA (2). In fact, one study reports severity of pain in knee joint OA to correlate better with the degree of synovitis and other soft tissue changes seen at arthroscopy than with the severity of the abnormality perceived radiographically (9). Thus undue reliance upon roentenographic data, leads one to believe that the problem

of diagnosis in OA may best be solved by studying the associated pathogenic factors (4,5,10). In addition, it appears that meaningful and comprehensive treatment of this disease is only possible if it stems from a reliable analysis of the normal and pathological stresses of the joint in question (5,6). In this regard, the role of muscle is of interest to several investigators (11,12,13,14).

For physical therapists, the evaluation and treatment of muscle is an essential component of the rehabilitation process. However, a major impediment to study on the effectiveness of exercise in treating OA patients has been the lack of basic knowledge concerning changes of muscle function in this disease. Therefore, characterisation of alterations which affect movement patterns and limb function in relationship to the aetiology and pathogenesis of OA may assist in defining the needs of the osteoarthritic patient in this respect.

The rationale for focusing specifically on the musculature in the development of OA is the fact that through it's effect on the distribution of load, muscles are a major factor determining the range of stresses placed on the joint. In particular, peak joint forces incurred during movement are dampened in part by eccentric muscular contraction (14,15,16). Failure to attenuate load is deleterious to articular cartilage and subchondral bone (16,17,18) and factors such as inadequate muscle capability

resulting from inappropriately timed and modulated levels of force production, may contribute to joint dysfunction that is of a mechanical nature (19,20,21,22).

It is also suggested that muscle imbalance may play a role in the development of joint dysfunction. Notably athletes with previous injuries and those requiring surgery, often present with agonist muscle weakness and associated tightness in reciprocal muscle groups. They may also display high rates of re-injury as a consequence (23).

In addition, persons with a history of muscle imbalance in the lumbo-sacral and cervico-thoracic region develop chronic pain syndromes and joint dysfunction (24). This suggests that the weaker or more imbalanced a muscle group is, the more prone the joint may be to injury.

Furthermore, with respect to OA of the knee joint (gonarthrosis), since both the frontal (valgus and varus) and rotational alignment (internal tibial torsion) of the lower limb are aetiological factors in the disease process, it is suggested that the nature of muscle activity as well as other influences of alignment must be considered when determining the genesis of the disease (25,26,27,28).

B: EVALUATION OF MUSCLE FUNCTION IN GONARTHROSIS

Information on the muscle property and its function in gonarthrosis is poorly documented. To date, such studies have largely been confined to both static and

dynamic torque measures and selected biomechanical parameters during gait (29,30,31,32,33). Several studies have accounted for the timing and level of associated force output of individual muscles of a given group (19,24,29,31). Fewer studies describe the role of muscle as an etiological factor in the development of the disease (13,21,22)

Although electromyography (EMG) has not been widely used to examine the muscle function with reference to OA, the role of EMG as an evaluative tool is well documented (34). In addition, measures such as EMG along with torque may reliably reflect the effects on muscle and the skeletal components (35). Although altered myoelectric activity can be demonstrated in the quadriceps femoris muscle of osteoarthritic knee joints (36), EMG has not been used to examine the associated force output or time factors related to tension development of the various components of the knee joint extensors in relation to gonarthrosis.

C: OBJECTIVES OF STUDY

The primary objective of this study was to quantitatively describe the pattern of myoelectric activity of the quadriceps femoris muscle in normal, "risk" and pathological knee joints during static and dynamic muscle contraction, with respect to evaluating the role of muscle imbalance in the natural history of gonarthrosis.

In addition, the study was designed to evaluate the relationship between time factors related to tension development in these three groups.

The objective was also to derive imformation pertinent to the evaluation and treatment of muscle dysfunction in gonarthrosis.

The specific questions posed were the following:

1.) Is the EMG pattern of the quadriceps muscle of normal females the same as the EMG pattern of females presenting with signs of tibial torsion or varus alignment?

2.) Is the EMG pattern of young females with tibial torsion or varus alignment the same as women with similar alignment who have osteoarthritis?

3.) Is there a relationship between time factors related to tension development in the quadriceps muscle and the presence or absence of tibial malalignment?

D: RESEARCH HYPOTHESIS

The research hypothesis stated that subjects with normal knee joints differ from subjects identified at "risk" or presenting with definitive OA of the knee joint with respect to the myoelectric activity of the anterior thigh muscle and to selected torque measures related to time.

E: SIGNIFICANCE OF STUDY

OA of the knee joint is a frequent cause of pain, discomfort, and disability varying in extent from mild to severe in middle-aged and elderly adults. Since the disease is thought to begin at a relatively early age, usually in the third decade, the demonstration of abnormalities in the presence of minimal clinical symptoms or perhaps even in their absence, is important with regard to preventitive as well as remedial measures (3).

While epidemiological studies have elucidated upon a number of factors associated with knee joint arthrosis, such as increasing age, trauma, sporting or occupational activities, these studies have not been helpful in establishing a causal role for pathogenetic factors in primary OA of this joint (4,5). In addition, most experimental and clinical studies have focussed on the later stage of OA development in which the gross cartilage fibrillation, bone remodeling and osteophyte formation occurring in the advanced stages of the disease, render the joint in a state much different from the pre-arthritic condition (5).

Thus articular cartilage or bone studies done on these grossly affected joints only offer hindsight into the problem. In contrast, a study of grossly normal joints and joints in a high risk population offers insight into the possibilities of determining signs that would allow

diagnosis and identification of the pre-arthritic joint before any gross changes arise (5).

Early detection of dysfunction, followed by appropiate intervention could reduce the risk of disability associated with OA.

To meet these goals, an improved ability to quantify any visible structural or functional changes in the limb segments afflicted by the pathology based on well defined and clinically significant variables is important. In addition, a carefully established normative base from a broad spectrum of the population is essential to define anthropometric and physiological variability.

In this regard, differences in the linear envelope detected full wave rectified EMG patterns during maximal voluntary contraction of the quadriceps femoris muscles in three groups of subjects were examined, thereby indicating the pattern of myoelectric activity in both normal and pathological knee joints with respect to the quadriceps femoris muscle under the conditions specified in the study. Time factors related to tension development were also studied.

F: OPERATIONAL DEFINITIONS

1. Electromyographic evaluation is defined as the use of surface or percutaneous electrodes to record

electrical activity from one or more muscles and to relate these recordings to corresponding body movements during attempts at a purposeful task or specified motion.

2. Full wave rectified EMG refers to the superposition of opposite phases of the motor unit action potentials such that all signals lie above the baseline.

3. Linear envelope detected EMG refers to the surface area delimited by the EMG signal after the full wave rectified signals are filtered.

4. Normalization is the use of a reference value by which comparison between levels of EMG activity between different muscles and different individuals may be calculated.

5. Muscle Imbalance implies either assymetry of the muscle function of the extremities when comparing ipsilateral and contra-lateral muscles or a differential within the normal values of opposing muscle groups within the extremity which is termed agonist-antagonist muscle imbalance.

6. Isometric contraction involves the development of force by an increase in intra muscular tension without any change in the length of the muscle.

7. Isokinetic contraction involves the development of muscle force during a concentric muscle contraction in which the angular velocity of the limb segment is held constant throughout the range of motion.

8. Torque is described as the product of a force that acts about an axis of rotation and it's perpendicular distance from the axis.

9. Gonarthrosis refers to osteoarthritis, degenerative arthritis, osteoarthrosis or degenerative disease of the knee joint.

10. The tibial angle is the angle formed in the frontal plane by the distal one third of the leg during stance with respect to the vertical axis of the femur. This angle, normally known as the valgus angle lies in the range of seven degrees plus or minus two degrees.

11. A varus knee is defined as having a tibio-femoral angle of 4 degrees of valgus or less (a neutral knee

is one ranging from 5 to 9 degrees of valgus).

12. Tibial torsion refers to the alignment of the tibia about its longitudinal axis, relative to the transverse axes of the proximal (knee) and distal (ankle) joint surfaces of the lower limb. This angle normally lies in the range of 15 to 25 degrees of external tibial torsion.

13. Internal tibial torsion refers to tibial alignment in which the normal range of external tibial torsion is decreased, usually causing the foot to lie adducted with respect to the tibia.

G: DELIMITATIONS

This study is delimited to :

1. The examination of healthy, "risk" and pathological knee joints in a selected female population.
2. The evaluation of the musculoskeletal status of the knee joints during voluntary muscle contraction in the sitting position.
3. The measurement of the motor activity over selected points of the quadriceps femoris musculature during isokinetic and isometric muscle contraction at selected speeds, using quantified EMG and surface

electrodes.

H:. LIMITATIONS

1. Reliability of EMG is limited to the calibration of the instrument, the ambient environmental conditions, filtering properties of electrodes and electrolytes, effective signal processing and conditioning, electrode size and placement and extent of subcutaneous tissues.
2. Reliability of torque measures is limited to the calibration of the Cybex II[1] Isokinetic Dynamometor.
3. The ability of each subject to exert maximal effort during muscle contraction is beyond the control of the researcher. Further, maxima of different subjects are likely to be different. This may influence the picture of muscle balance imbalance.
4. The osteoarthritic and aging changes can not be discriminated among patients.

---

[1]Cybex. A division of Lumex Inc. 2100 Smithtown Avenue. Ronkonoma, N.y. 11779

II.REVIEW OF THE LITERATURE

A. INTRODUCTION

The gross morphological changes in and about joints in patients with OA were described by the Hunter brothers over 200 years ago (37). In 1802, Heberden described the firm nodular swelling of the distal interphalangeal joints that bear his name and osteoarthritic changes of the proximal interphalangeal joints were described by Bouchard in 1884 (37). In general however, throughout the 19th and much of the 20th century, OA was not distinguished from other arthritic conditions, particularly from rheumatoid arthritis (RA). In 1904 however, Goldthwait classified OA as atrophic and RA as hypertrophic arthritis and in 1907 Garrod related the earlier findings of Heberden and Bouchard to osteoarthritic involvement of the proximal and distal interphalangeal joints (38). In recent decades, attempts have been made to subdivide OA into specific categories and Collins (39) introduced a classification system upon which much of the current metabolic, biochemical and immunological studies are based. Kellgren (40) made a significant contribution in this regard and classified subjects into those with localised and those with generalised OA. More recently the American Rheumatism Society has sought to establish a classification system for the diagnosis and reporting of OA (41).

This review will describe the aetiology and pathogenesis of OA with emphasis on the role of muscular changes that accompany the pathological changes with respect to the knee joint. Test procedures such as EMG and isokinetic dynamometry will also be discussed. Physical therapy treatment approaches in the management of OA will also be described.

Ideally this literature search should include clinical application of biomechanical and neurophysiological concepts with respect to OA and the physical management thereof. However, a paucity of imformation limits such an endeavour and reflects need for the application of dynamic principles of joint function to the understanding of this disease.

B: OSTEOARTHRITIS

OA is principally a disease of the articular cartilage that covers the ends of bones within diarthrodial joints (42). It is the most prevelant of the rheumatic joint diseases and one of the most common of the chronic diseases affecting mankind (1).

The disease is slowly progressive, and characterised clinically by pain, deformity, and limitation of motion, particularly in the hands and the large weight-bearing joints of the lower limb (43). While the disease has no known cause the main risk factors in OA are well known and

are substantiated by epidemiological studies. They comprise extrinsic factors, essentially mechanical stresses and intrinsic factors especially aging (39,43,44,45). Some authors concur with the view that OA is analagous to "heart failure". That is, it is a disease of an organ, namely the "diarthrodial joint" which can result from a number of different and interrelated factors and is not merely a disease of a single tissue, ie., the articular cartilage (3,40).

Traditionally, OA is classified as primary or idiopathic if a cause is not apparent and secondary if one is (43). Although these two categories may appear to divide OA into two distinct subsets or types, more recent studies indicate that primary OA is often related to minor abnormalities of the underlying joint which escape detection. Harris (47) concluded from a study of the disease in the hip joint, that the term "primary" applied to less than 40 percent of subjects diagnosed as such and that with improved detection methods, it is probable that all OA will be eventually be reclassified and recognised as secondary.

The likelihood is high that OA is a disorder that is non-linear in it's evolution; that is, it progresses slowly at different rates in different people over a prolonged asymptomatic period. Then in some, the disease becomes clinically symptomatic, at which time there is rapid

exponential progression to an unrelenting state of disability perhaps as a result of secondary imflammation. This point in time, may vary considerably from joint to joint and from individual to individual. While radiographic findings are usually diagnostic, to date no correlative data have established radiographic or histological markers to coincide with this event. The initial changes in an osteoarthritic joint are still the subject of speculation (40). The clinical, laboratory and radiographic criteria for the diagnosis of clinical or definitive OA are listed in Appendix A (41,48).

CLASSIFICATION

The anatomical patterns of OA suggest this is not a single disorder but rather a large heterogeneous group of conditions (49). For instance polyarticular small joint OA, usually presenting as a nodular arthritis of the metacarpophalangeal joints, has a distinct age, sex and race distribution which sets it apart from large joint pauciarticular OA which is common in the knees and hips. Furthermore, pauciarticular large joint OA can be subdivided into hypertrophic (well-marked new bone formation and remodelling) and atrophic (minimal new bone formation) types in which the distribution of the two groups differ significantly. In addition, generalised OA is said to be a combination of polyarticular small joint and

pauciarticular large joint disease, OA with skeletal hyperostosis is also found (50).

In terms of pathology, clinical and distinguishing characteristics, OA disorders may be differentiated into three basic patterns. Type I lesions exhibit progressive deterioration of previously normal cartilage due to excessive stress at one point of the joint (absolute overload). Type II lesions occur due to an intrinsic defect which renders the cartilage susceptible to normal levels of stress (relative overload). Type III lesions arise where these two mechanisms co-exist and may occur in such conditions as chondrocalcinosis (50). Radiographic representation of population groups further classify the disease into four distinct grades (appendix A).

GENERAL PREVALENCE

The prevalence of OA in relation to age, appears the most consistent feature of the disease. In any population over 35 years of age, OA can be detected in one to two individuals out of three, and the severity of OA grades three and four has been found to increase with advancing age (51). One suggestion for this exponential rise has been explained in terms of a mutation in the recognition of autologous cartilage proteins by cells of the immune system with aging, which due to their isolation from the blood system are viewed as "foreign" (52). A second explanation

is the finding that increasing secondary mechanical instability superimposes itself on the aging cartilage over a long period of time (52). Forman and associates (53) noted that OA will not inevitably occur in the aged, nor when present was it certain to progress and suggested that factors other than aging may play a role in the aetiology and pathogenesis of OA.

The specific joints affected by OA vary depending on the age of the groups studied. Peyron (51) reports the disease is seen earliest at the age of 25 in the metatarso-phalangeal joint. At the age of 35, OA begins in the wrist and at age 45, the distal phalangeal joint of the index finger, the carpo-metacarpal, metacarpophalangeal and proximal phalangeal joints may all develop OA. However, these data may not include injuries and subsequent joint damage incurred by very young athletes, as described by Michieli and associates (54).

Up to the age of 45 years, OA is slightly more prevelant in males than females. After age 45, the disease becomes more severe in females, with multiple joint involvment in 50 percent of females over age 55 in contrast to 29 percent of males at the same age. In females, the disease is primarily localised to the distal interphalangeal, first carpo-metacarpal and knee joints, while the most frequent pattern in the male involves the metacarpo-phalangeal and hip joints (3).

NATURAL HISTORY

The gross lesions of OA culminate in a complete loss of articular cartilage with exposure of bone, eburnation, remodelling and osteophytosis (42,55). However, the anatomic definition of OA by examination of gross pathology cannot be used in longitudinal clinical studies of OA, therefore necessitating the use of clinical and radiographic criteria (41,48). While osteophytes are the major or even sole criterion used in many studies, Danielsson and Hernborg (56) found that only one third of patients with osteophytes alone progressed to joint space narrowing. In a second study reported by Hernberg and Nilsson (57) only 34 of 86 patients with osteophytes developed other radiographically apparent structural changes over a 10 year period. Hernborg and Nilsson (58) subsequently followed 71 patients with knee OA for 10 to 18 years to assess rate of progress of OA. In this prospective study, symptoms improved in 15, remained unchanged in 23 and worsened in 49. Of note was the finding that the OA changes remained limited to the compartments initially affected. Additionally, varus deformity predominated over valgus deformity and was associated with joint instability and a poor prognosis.

Joint deterioration due to OA is a relatively slow process. Although few longitudinal studies have been described, Plato and Norris (59) reported the maximal rate

of degeneration in the distal interphalangeal joints was the equivalent of about one grade per individual over an interval of 12 to 16 years. In addition, an even lower rate of degeneration was found with respect to the proximal interphalangeal joints.

Sokoloff (60) studying the natural history of degenerative joint disease in small laboratory animals noted conspicuous changes in the soft tissues of the joints in the presence of joint degeneration. Similarly Videman and associates (61), using an animal model, found changes in the ligaments, joint capsules, and menisci in their study of the osteoarthritic joint. In the animal models studied by Sokoloff (60), the knee was found to be the most severely affected joint, with medial compartment dysfunction generally greater than lateral compartment dysfunction. In addition, at times, the patella appeared to be displaced medially, while the tibia was found internally rotated on the femur.

Ogata et al (25), produced unicompartmental OA by applying varus stress to the moving rabbit knee joint and noted degenerative changes confined to the medial tibial and medial femoral articular surfaces. In particular, within the range of varus stress used, duration appeared to be more important than magnitude of varus stress in determining the severity of cartilage damaged.

Although most patients with OA of the knee are found

to present with a varus deformity, some may present with valgus deformity (26). It is not clear however, to what extent mechanical factors dictate the localisation of the disease or it's primary progression to an angular deformity (26), although it seems likely that it is primarily the tibial tilting angle to the floor that determines the joint forces in the deformed knee (62). In particular, Minns (195) reported a tibiofemoral angle of seven degrees varus to place a higher load on the medial side of the knee and indicated that concomitant high values of medial subluxation may predispose the joint to the degenerative changes seen in gonarthrosis. Ebong (64) commented that although minor degrees of genu valgum are not neccesarily related to OA, severe degrees of deformity are related. In addition, both valgus and varus deformities are known to be associated with a greater progression of degenerative changes than is found to occur in knees with a neutral angulation (56,57). Kostiuk and associates (65) found marked change in the amount of weight borne by the medial and lateral compartments of the knee in the presence of only three degrees of valgus or varus deviation.

With regard to tibial torsion, Yagi and Sasaki (28), found the degree of external tibial torsion decreased as the stage of the disease advanced.

MECHANICAL FACTORS

The concept that primary OA is a "wear and tear" process has not been successfully substantiated. Since the coefficient of friction in joints has been measured experimentally and found to be remarkably low (66) it is inconceivable that joints could wear out by simple rubbing (16). Radin and associates (16) also report that joints will not wear out even when the considerable lubricating advantages of synovial fluid have been removed from the lubricating system. However, unlike rubbing, the forces the joint is subject to from longitudinal loading are reported to be quite substantial and come not from weight bearing per se, but from the contracture of muscles which span the joint.

The frequency and type of knee movements for specific activities such as walking, ascending and descending stairs and sitting have revealed a high rate of repetitive acts in normal persons performing these common daily activities (68). Experimental findings of Radin and others emphasise the role of repetitive loading in joint wear in this regard (12,13,16). Folman and co-workers (69) report that rapid cyclic forces may cause fatigue failure and gradual tearing of fibers in young cartilage and more so in aging cartilage. With regard to mechanical force, normal cartilage may also fail as a result of a single mechanical event as demonstrated by Unsworth (44). Bennett and

associates (70) reported on age changes in the knee joint and concluded that shearing and twisting stresses applied to the weightbearing surfaces of the tibial condyles produced the earliest signs of articular change.

Optimal distribution of load across a joint is paramount to maintaining joint integrity, and Bullough (45) states that consideration must be given to both the structural components as well as physiological mechanisms of joint function in determining vulnerability to load. In particular, since the diarthrodial joint moves normally only when the actions of the skeletal muscles that act across the joint are physiological, any discussion of the origins and failure of hyaline articular cartilage in OA must take into account the role played by skeletal muscle and the neural control mechanisms thereof (45). This concept is supported by Radin (15) who reported evidence of widespread neuromuscular dysfunction in generalised OA and by Janda (24) who found a high incidence of degenerative joint changes associated with problems of neuromuscular origin arising in early childhood. Farfan (71) stated that the normal physiological behaviour of a joint was dependent upon control mechanisms involving the sensory, central nervous and muscular systems. Additionally altered mechanoreceptor function is postulated to result in excess muscle tone which by increasing the load on the joint could lead to cartilage breakdown. In this regard, Moskowitz

reported the work of Palmoski and associates, who noted that sectioning of the posterior and medial articular nerves of the knees of normal dogs led to an increase in cartilage water content, proteoglycan synthesis and extractibility, findings similar to those occurring in definitive OA.

In regard to the aetiology of Heberdens nodes in generalized OA (GOA), Smythe (72) postulated the involvement of the musculature whereby following an early fixation of the extensor tendon at the proximal interphalangeal joint, compensatory distal interphalangeal joint flexion was produced by the powerful flexor muscles. Compressive forces acting on the cartilage and subchondral bone were increased as a result and a crush injury similar to the subchondral fractures described by Radin (16) developed following excessive use. In support of this concept, Buckland and associates (74) examining the relationship between soft and hard tissue changes in nodal GOA, both clinically and using microfocal radiography, noted that the interrelationship of osteophytes and the overlying soft tissue nodes correlated with the mechanical forces occurring in normal hand function .

Additionally, the frequent association of unicompartment arthritis of the knee with angulation deformities emphasises the role of mechanical factors in the proposed aetiology of the disease (25,26,27,28).

MUSCULAR REFLEX MECHANISMS

Neuromuscular reflexes play an important role with respect to maintaining structural integrity. Carlsoo and Johannson (75) showed that when subjects fall to the ground on the outstretched hand all the muscles surrounding the elbow joint are "strongly activated some tenths of seconds before the hand touches the surface". Consequently the musculature is prepared to protect the joint. Similarly, in landing from a voluntary jump, deceleration is brought about by an accurately timed burst of neuromuscular activity which appears to be preprogrammed well in advance of the actual fall thus absorbing the energy and load of the impact (19). In contrast, it has been demonstrated that an unexpected fall is associated with a very significant jolt and the load of impact is poorly attenuated (19,76). Joints will also develop degenerative changes in response to load, if muscle function is inhibited (20). These findings indicate that while appropiate muscular responses may protect a joint from deleterious loading effects, failure of this mechanism may lead to the onset of degenerative joint changes (11,12). In particular, the shock absorbing mechanism involving the stretching of the muscles under tension have visco-elastic time-dependent mechanical responses, which are relatively ineffective in

withstanding sudden impulse loads of short (50 msecs.) duration. In contrast, loads of 500msecs. duration, with long rise times are found to cause no significant joint destruction (14). Inter-individual variations in fitness levels, muscular development and reflex responsiveness may modify this response and older less active individuals are more likely to be susceptible to joint damage as a result (78).

The work by Radin and associates (15,16,18) suggests that muscle must be capable of well timed and appropiately modulated contractions. In contrast, in abscence of the optimal amount and rate of development of muscle tension, joint destruction would almost be assured.

MUSCLE PATHOLOGY

Richards (78), reviewed the functional role of muscle fibre type reporting on the work of Sherrington and co-workers who found that muscle fibres are organised into functional units of motor control within a muscle. Furthermore, muscle fibres in these motor units interdigitate with other muscle fibres from other motor units within a muscle to form a mosaic of fibre types in cross-section. Fibres with long contraction times are differentiated from those with shorter contraction time (79). In general, motor units are broadly classified as fast-twitch, glycolitic, high tension, type II units, while

type I units are slow-twitch, oxidative, low tension units. Type II units are further classified as type IIA,IIB and IIC fibres on the basis of contraction speed and rate of fatigue (80).

Muscles high in fast twitch fibre content demonstrate higher peak torque values, and a greater susceptibility to fatigue than muscles composed mainly of slow twitch fibres (128). It has therefore been suggested that muscle fibre type or predominance may predispose an individual to cartilage damage. For example, in an individual with a low proportion of slow-twitch (red) muscle units, an inability to perform tasks of endurance may render the cartilage more susceptible to injury. Conversely, an individual with a low proportion of fast-twitch units, may display a decreased capacity to produce a fast rise in tension in response to sudden loads (80). Motor unit discharge characteristics in aged persons (70 to 79) are found to discharge more slowly than those in younger groups and failure to develop adequate levels of tension in response to intermittent impact, may explain the high incidence of joint degeneration found in this age group (81).

Sirca and associates (82) have found a preferential loss of type II fibres in the glutei of human subjects presenting with hip joint arthrosis, while Martin et al (83) note similar type II fibres loss of the vastus lateralis muscle in persons with OA of the knee joint

indicating the possible influence of fast twitch fibre loss in the disease process.

However, although muscle in OA is generally associated with type II fibre atrophy (82,83), Sirca and Susec-Michieli (82) found a decreased fibre diameter for both type I and II fibres in the gluteus medius and tensor fascia lata muscles in younger patients with OA and a recent study by Glasberg and co-workers (84) has shown more extensive morphological changes in the quadriceps femoris muscles of persons with knee joint arthrosis than those previously reported. In particular, denervation atrophy and myopathic changes with a positive atrophy factor in both Type I, Type IIA and Type IIB myofibres were noted. These results indicated that patients with severe gonarthrosis may present with significant myopathy as well as neuropathic changes. These changes are notably different from previously reported results of muscle biopsy in patients presenting with rheumatoid arthritis, femoropatellar arthritis, meniscus tears or dislocating patellae. Whether muscle pathology is a predisposing factor in the disease process or merely a secondary phenomonon arising from disuse subject to the onset of gonarthrosis is not known (84). However, Harding (85), noted disuse atrophy occurring as a secondary phenomonen in arthritis. Since static and dynamic torque measures performed in conjunction with biopsy in elite athletes show

a high correlation between fibre type and strength measures (86), altered strength measures noted in subjects with OA of the knee joint (32,87), as well as muscle fibre changes (84) likely emphasise the extent of muscle pathology in OA.

MUSCLE IMBALANCE

Factors affecting muscle tone are found to result in typical reactions or patterns of muscular agonist-antagonist imbalance. Muscle imbalance may produce aberrant movement patterns, increasing abnormal joint stress, thereby permitting undue joint loading. It is speculated that an increased incidence of joint injury and the development of degenerative joint changes may arise as a consequence (23,24).

A clinically developed muscle imbalance is partially the result of reflex mechanisms. While certain muscles respond to a given situation e.g. pain by tightness and shortness, others react by inhibition, atrophy and weakness (24). Muscle tightness or spasm occurring secondarily to injury or disease may generate substantial compression on joint surfaces as noted by Janda (24) and De Bont and associates (88). Arsever and co-workers (89) found muscle weakness produced by myectomy to result in the onset of OA. However, both a paralysed limb as well as the remaining joints of an amputated limb are spared the affects of OA

(90,91). Others note that suboptimal loading of a joint from inadequate contraction of the muscles that span the joint, may significantly alter the viability of the articular cartilage (67,72).

In hip joint arthritis, muscle lengthening or sectioning procedures such as the "hanging hip", result in movement of the major weight-bearing site on the femoral head, eventually leading to cartilage remodelling (92). In contrast, Bullough (93) suggests differing locations of wear in OA femoral heads noted in autopsy studies result from abnormal muscle "pull," suggesting the possibility of a "clenched" hip syndrome analogous to "clenching" described in mandibular arthritis (88).

The role of muscle imbalance in the development of gonarthrosis is unclear. However, Mariano and Caruso (94) found vastus medialis and lateralis imbalance with respect to patello-femoral arthritis. In addition, Merrifield and Kukulka (95) monitoring the EMG activity of the quadriceps femoris muscle with respect to varus and valgus deviation, noted vastus medialis activity to exceed that of the vastus lateralis muscle during valgus stress. In contrast vastus lateralis activity increased with varus stress, suggesting muscle imbalance may be a factor in knee joint malalignment. However there is no definitive work which can clarify the cause and effect relationship between muscle imbalance and OA of the knee joint.

Identification of neuromuscular alterations associated with OA would enhance the possibility of influencing the disease process. Since mechanical factors are a major factor in the onset of OA, the importance of these factors associated with load attenuation should be emphasised (96).

In summary, current evidence suggests that a subject risks joint damage in relation to repetitive impulse loading if :

1) his muscles are not strong or co-ordinated enough to attenuate the shock of loaded joints during activity

or

2) the subject routinely engages in activities which demand a faster reaction time than his/her neuromuscular system is capable of (97). Although poorly documented, it is likely that collison sports in particular would be implicated in this regard.

C. THE KNEE JOINT AND OSTEOARTHRITIS

The knee is the most commonly involved major joint affected by OA and a frequent source of functional disability and pain (98,99). In addition, excess mortality and decreased survival rates have been noted in women age 55 to 74, who present with radiographic changes of OA of the knee joint compared to those women with normal radiographs (98). Most radiographic cross-sectional surveys

have detected an increasing prevelance of knee OA with age (101,102). This is confirmed by autopsy studies of knee pathology (43,44). In contrast, Forman et al (103), performed a cross-sectional survey 682 elderly ambulatory and hospitalised patients and found no significant change in prevelance or severity of gonarthrosis to occur with increasing age. Kaplan (104) concluded that gonarthrosis is a specific disease occurring in only a proportion of the population and may not necessarily be progressive. With the exception of obese individuals, Berkhout and associates (105) found improvement irrespective of age and duration of knee complaints in a six month retrospective study of 72 patients with OA of the knee joint.

Like structural changes, the prevelance of knee pain increases with age (63). However there is a discrepancy between the amount of significant daily pain present in patients surveyed by the United States National Center for Health Statistics and the prevelance of radiographic degenerative joint disease of the knee. A substantial portion of the younger patients have knee pain but no or minimal radiographic OA. While this discrepency might be indicative of early OA, this postulate remains untested because of lack of adequate noninvasive diagnostic techniques (55). Parnell (106) suggested that the presence of knee effusion in healthy young adults, found to increase in prevelance with age, weight and bicondylar femoral

width, was likely indicative of early degenerative arthritis.

NORMAL KNEE JOINT STRUCTURE AND FUNCTION

Bony Structure

The knee is classified as a hinge joint. It is a tricompartmental joint with considerable surface area and incongruity. The osseus portions of the knee include the femur, tibia, patella and fibula which form two distinct articulations lying within a single capsule. These are the tibiofemoral joint and the patellofemoral joints (107,108). The femur is found to slant relatively medially upon a vertical tibia, forming an angle of approximately 10 to 15 degrees between the vertical axis of the two bones. With reference to the frontal plane, this angulation is known as the physiological valgus angle (107,108). In general, the medial tibio-femoral joint bears 60 percent of the transmitted load, while the lateral compartment carries the remainder (77).

Joint movement is considered a relative sliding motion of the opposing condyles. In the last 10 to 20 degrees of extension, however, the femoral condyles roll forwards slightly on the tibia. Since the radius of curvature of the femoral condyles decreases from front to back, the medio-lateral axis of the joint varies in position, depending on the angle of flexion (109). Early

and tibia during movement indicated that movement occured predominantly on the medial side of the joint when the knee was in 0 to 20 degrees of flexion (110). In active or passive extension of the knee joint, Hallen and Lindahl (111) found that about seven degrees of outward rotation of the tibia was combined with sagittal plane movement to lock the knee in extension.

During weightbearing, the knee is stabilised by ligamentous and musular components. The most important of these are the anterior and posterior cruciate ligaments, the medial and lateral collateral ligaments and the quadriceps femoris and hamstring musculature.

Articular Cartilage

Structure

The articulating surfaces of the knee joint are lined by articular cartilage. Articular cartilage is a highly organized tissue, relying on finely tuned metabolic processes for continued functional integrity (112). Cells known as chondrocytes occupy one to ten percent of the cartilage volume and the remainder is an extracellular matrix composed of collagen fibres, proteoglycan molecules, water (65% to 75%) and small amounts of lipid and inorganic material (38,113).

Collagen fibres comprising about half the dry weight of adult articular cartilage and are anchored to the

subchondral bone and tend to run vertically into more superficial cartilage. At the articular surface, collagen fibres are aligned tangentially. This arrangement ensures that the deep cartilage can resist compression and the superficial fibers can resist friction (113,115). The collagen network constitutes a radial array of collagen fibrils which are cross-linked along their length to facilitate dimensional stability in the unloaded state and to provide mechanical resilience under a wide range of loading condition (114).

Abundant organic substance in cartilage are proteoglycan aggregates. These are molecules consisting of a long hyaluronic acid backbone to which many proteoglycan subunits are attached. These subunits contain numerous side-chains of chondroiten and keratin sulphate and carry a large fixed negative charge at neutral pH. This charge creates an electrostatic repulsion that forces these macromolecules into an extended conformation within the three dimensional collagen network to become effectively immobilised. Furthermore this negative charge causes the proteoglycan aggregates to occupy a large solvent domain in aqueous solutions. A "swelling" pressure is therefore generated within the cartilage matrix, and the constraint to this swelling given by the collagen network results in a mechanically taut composite structure capable of sustaining compressive loads (114).

compressive loads (114).

Chondrocytes constitute the cellular element of articular cartilage and are responsible for their continual degradation and replacement of cartilage matrix. Surface cells are more numerous and assume flattened and discoidal shapes and are considered quiescent, while the deeper cells are larger, more spherical and actively synthesise protein (116,117,118).

In the knee joint, the medial tibial condyle has a biconcave oval superior surface. The articular cartilage lining this region is relatively thick and provides for a more stable articulation than the lateral tibial condyle (108,109).

The thicknesses of the articular cartilage measured at the centres of the contact areas from 1.8 to 3.3 mm. and are found to be greater at the contact areas of 15 and 30 degrees, than at 60, 90 and 120 degrees of contact. For the lateral condyles, the thickness on the tibia is greater than on the femur by an average of 30 percent, but on the medial condyles the thickness is closely equal (55).

Function of Articular cartilage

Articular cartilage is an avascular tissue and there is little evidence that any significant diffusion of nutrients takes place from bone into mature cartilage (118). It is likely therefore, that synovial fluid is

almost entirely responsible for cartilage nutrition. Radiolabelled nutrients within synovial fluid diffuse rapidly into cartilage, while activity, which increases the circulation of the fluid, helps to maintain a maximum concentration of nutrients on the articular cartilage surface (118).

Articular cartilage is highly deformable and has a combination of elastic and visco-elastic properties. When load is applied, there is an elastic deformation and with time there is a slow increase in deformation known as creep. With the release of load, there is an initial elastic recoil, then a slow recovery creep back to the preload configuration. The flow of water through the matrix, the rate of load application, and the thickness of the cartilage, determine to a great extent the shape of the deformation curve for articular cartilage (119). Various factors affect the contact of the load bearing areas of a joint with respect to articular cartilage. The area of contact will depend upon the degree of elastic and visco-elastic defromation of articular cartilage layers, and the magnitude of the load, as well as the duration of the load will be two major variables, in addition. In general, the forces normally acting on the cartilage must fall within the range suited to the continued vitality and viability of the cartilage cells (44). That is, they must be of sufficient amplitude to provide for the nutritional supply

from the chondrocyte matrix but must not be excessive as to produce necrosis. Where stress on the cartilage falls outside the optimum range, degenerative processes may ensue (4).

Pathology of articular cartilage

The gross lesions of OA culminate in a complete loss of cartilage with exposure of bone, eburnation, remodelling and osteophytosis (9,42), while the most characteristic microscopic picture is one of fibrillation of the superficial layer of cartilage. Here, shredding and small breaks in the surface can be seen. The lesions are initially tangential to the surface but become more vertical with time, forming clefts which penetrate the cartilage. This results in ulceration and gross loss of cartilage material which may extend toward the region of the subchondral bone (4,60).

In all forms of OA, biochemical changes are detected. In general, the proteoglycan aggregates exhibit increased extractibility despite their excessive production (100) and the units are smaller than normal. Loss of the proteoglycan content leads to a decrease in compressive stiffness and elasticity within the cartilage and an increase in hydraulic permeability (121) and the cartilage is rendered more vulnerable to mechanical stress as a consequence (122).

Studies of cartilage collagen show that whereas the overall content may increase with the severity of the disease (123), the structural quality of the collagen my become altered whereby the tensile strength of individual collagen fibres may be reduced (124). In addition, at any given point in time, collagen synthesis begins to fall as the chondrocytes become inactive (125.)

A relationship between changes in the cartilagenous and subjacent bony tissues exists whereby there is generally increased bone formation, thickening of trabeculae, sclerosis and cyst formation (126). In addition, tne synovial membrane may proliferate to form a pannus and produce a synovial effusion (127).

The morphological differences found between normal aged and osteoarthritic cartilage suggest that factors other than aging are involved in the genesis of OA (117).

The Knee Joint Musculature.

Although the joint is the largest in the body it is also one of the most unstable and is frequently subject to injury as a result. While knee stability is provided by the complex interaction of a multitude of factors including ligaments and other soft tissue restraints, active muscular control serves to distribute the load in an optimum manner. The mobility requirement is provided primarily by the knee joint musculature amongst other factors (128,129,130).

The musculo-tendinous units of the thigh are generally separated into two synergistic groups, namely the quadriceps femoris and hamstring muscles. The quadriceps femoris (QF) muscles are formed by four components that extend the knee and assist in hip flexion. The four named parts are the vastus medialis (VM), the vastus lateralis (VL), vastus intermedius (VI) and rectus femoris (RF) muscles. Of these muscles, the three vasti take their origin from the femur, while the rectus femoris originates from the ilium. The RF inserts into the tibial tubercle via the patella and ligamentum patellae and the VI inserts into the tendon of the RF. The VM and VL muscles insert into the tendon of rectus femoris on each side as well as the respective sides of the patella and the knee joint capsule (129). Since patellar tendon moment arms (distance between point of force application and axis of rotation) are shorter in women, accounting for the development of 20 percent greater knee joint forces than men for the equivalent extending muscular moment, stresses on the cartilage as a result of compressive forces will be greater. It is suggested that overweight women in particular, are therefore exposed to higher joint stresses than men of corresponding weight thereby explaining the observed vulnerability of overweight women to developing gonarthrosis (131).

The hamstring muscles are primarily involved in knee

joint flexion (130). Other muscles associated with knee joint function are the tensor fascia lata, sartorius, gracilis, gastrocnemius, popliteus and plantaris muscles (129). Of the muscles which assist in providing stability, the quadriceps femoris muscle is the most important. The quadriceps muscle also plays an important role in gait by extending the knee joint and assisting in the deceleration of the forward motion of the femur on the tibia (128,129). In addition, measures of abduction and adduction of the tibia with the thigh muscles in both relaxed and contracted states, indicate the stabilizing influence of the contracting quadriceps and hamstring muscles in this regard (132).

Biomechanics

The force exerted on the knee in standing with symmetrical support on both feet is half the body weight minus both lower legs and feet. During one-legged stance, the knee supports the partial body weight (body weight minus the loaded lower leg and foot). This force acts first medially and behind the knee, then medially, and finally medially and in front of the knee. This force is balanced by muscular forces which act first laterally and in front, then laterally and behind the joint. As a result, the force of the body mass normally acts on the centre of gravity of the weight bearing surfaces, evoking compressive stresses

which are evenly distributed over both tibial plateaus (133).

During gait, the knee is subjected to static and dynamic forces developed by the mass of the same part of the body and to both muscular and ligamentous forces. At heelstrike and just before toe-off, when the maximum joint load occurs, there are large varus moments acting at thjoint and the medial condyle is found to carry more load than the lateral (134). The static and dynamic distribution of force is altered if the lateral muscular stay is weakened and the body weight acting on the knee is then increased medially (133).

In this regard, clinical evidence suggests that in some individuals, obesity outstrips the ability of the lateral muscular stay to counter-balance body weight. In addition, medial displacement can be the consequence of leg length discrepency resulting from the shifting of the centre of gravity of the body. In an attempt to get the feet under the centre of gravity, subjects may walk slightly bow-legged and develop a varus deformity, significantly increasing the stress on the medial compartment. Preferential arthritic involvment of the medial side of the knee joint in patients who are obese, supports the liklihood of this theory (18,133).

Moskowitz (135) reports on a study in which joint responses to alterations in load bearing were studied

following valgus angular osteotomy of the tibia. Animals subjected to wedge osteotomy were observed for up to 3 months after surgery. In all instances histological evidence of degenerative abnormalities were present. In particular these changes were limited to the lateral tibial and lateral femoral condyles. However, a study of static and dynamic loading patterns by Harrington (136) does not support the notion of a direct relationship between angulation deformity and the distribution of load in the knee, although knees with a varus deformity are found to show a more predictable loading pattern than knees with a valgus deformity. Kettlekamp and Chao (81) suggest that the proportion of total load is shared by the medial and lateral plateaus such that the medial compartment transmits most of the force when a varus deformity is present, as do the lateral condyles when a valgus angulation exists.

Muscle Imbalance

The role of muscle imbalance in the development of knee joint dysfunction remains obscure. In general however, tight muscles may act in an inhibitory way on weakened muscles and thereby alter joint function (24).

The hamstring muscles are mainly involved in brisk non-weightbearing activities, while the muscles such as the vasti and more particularly vastus medialis rely heavily on sensory feedback for their action especially during weight

bearing (138). Arthrokinetic reflexs reinforce and co-ordinate flexion-extension movements of the leg. For example, if the flexors of the thigh start to contract either as a result of voluntary or reflex mechanisms, these reflexes will inhibit the thigh extensors and increase the excitability of the flexors (131). Thus tightness of the hamstring muscles may result in inhibition of the quadriceps muscle. Loss of knee extension imposes a strain on the knee joint both in stance and during the swing phase of gait in particular (31).

Systematic observations of arthritic knee joints show that knee flexion contractures are a characteristic feature of these joints, supporting the concept of hamstring muscle tightness in the pathogenesis of gonarthrosis. Stauffer et al (31) noted that during stance, the average range of knee flexion in 65 subjects with gonarthrosis was approximately 9.2 degrees. In addition these workers found a significant inverse correlation ($p < .05$) between standing flexion and muscle strength. This finding has been substantiated by several other investigators (29,32,33).

Miller and associates (99) found the degree of knee flexion correlated with increased severity of arthritis involving the medial joint compartment as well as the patellofemoral joint. In addition, Miller et al (99) attributed the greater frequency of cysts noted in knees with flexion deformity to an increased load per unit area

resulting from this deformity.

Waugh, Newton and Tew, (140) conducting autopsy studies of arthritic knee joints, noted, that in almost all cases, lesions of the medial and lateral femoral condyles were associated with a knee flexion contracture. These workers inferred, that the development of flexion contractures might precede the onset of an arthritic lesion and any knee which does not fully extend is likely to develop pathological changes. In this regard, flexion deformity of the knee increases the force per unit area, since the area of contact between the tibia, meniscii and femur decrease with flexion. Thus a fixed flexion deformity produces loads carried across a smaller surface. This is compounded by increased quadriceps action required to maintain stability in a flexed position. The net result is increased stress across the knee (24).

Michieli and associates (154) suggest that during the time of the adolescent growth spurt enhanced bony development around the knee may produce significant alterations in the activity of adjacent musculature thereby predisposing the joint to OA in the long term.

C: EVALUATION of OA

While clinical criteria for the diagnosis of idiopathic OA of the knee joint have been established (41) radiographic evaluation and bone scintiscanning remain the

most important tools employed in the diagnosis of OA. This approach is not always sufficient in helping clarify the diagnosis in early OA. This statement is also true with regard to the use of ultrasonography, ferrography, biopsy, measures of intra-venous osseous pressure and various laboratory tests, although arthroscopic examination, synovectomy and magnetic resonance imaging may prove useful in some instances (141,142,143,144).

In contrast, preliminary studies in the use of acoustical recognition techniques and the accelerometor in particular, show promise with respect to the use of non-invasive techniques in identifying early signs of joint dysfunction (11,12,13).

With regard to the clinical evaluation of the knee joint, electromyography, torque measures, force plate analyses, oxygen consumption and energy costs as well as gait analyses and functional status parameters have been studied (29 30,31,32,36).

A. Pertinent Studies

Strength Measurements

Muscle strength measured isometrically by Wigren and co-workers (147), showed no significant difference between the non-diseased side of subjects with unilateral gonarthrosis and the knees of healthy volunteers. However, significantly less strength could be noted with respect to the osteoarthritic knee joint. In contrast, Nicolas,

Strizak and Veras (148) in a study of thigh muscle function in different pathological conditions of the lower extremity, noted that subjects with moderate to severe degenerative arthritic knee changes, showed no significant muscle group weakness with respect to five major muscle groups of the lower limb. That is, in all of the muscle groups tested, none showed a consistantly significant deficit from the unaffected or control leg. However, the mean total leg strength in the affected knees did display an 18 percent deficit with respect to normal leg muscle strength.

TABLE 2.2: Thigh Muscle Weakness in different pathological conditions. Adapted from (148).

Group	Number	Mean strength		% deficit	significance
		affected	control		
Ankle	14	316	425	26	yes
Back	19	341	380	10	yes
Instability	23	382	444	14	yes
Patella	16	341	386	12	yes
Arthritis	5	299	363	18	no

Nordesjo et al (30) studying the isometric strength in patients with OA of the knee joints noted a 55 to 70

70 percent reduction in flexor and extensor muscle strength compared with a group of healthy volunteers, supporting the work of Wigren and co-workers (147). In this study the percentage decline in strength was greater for females with respect to the quadriceps femoris muscle and greater for males with respect to the hamstring muscle. Similarly, an isokinetic study of peak torque activity for both knee flexion and extension at four speeds reported by Lankhorst and associates (32) showed readings taken from OA limbs to be 65 to 83 percent lower than those of the normal limb.

Beals and associates (87), found that with respect to isotonic leg flexion and extension, strength was diminished in OA subjects compared to age and sex matched healthy controls but noted no significant differences in the maximum torque in isometric extension with the knee positioned at 90 or 45 degrees.

These result suggest that subjects with gonarthrosis have decreased isometric muscle strength compared to age matched healthy subjects.

Electromyographic Studies

EMG recordings from the quadriceps muscle during attempted maximal contraction have been shown to be significantly altered in gonarthrosis (151).

In particular, Brucini et al (36) noted the presence of involuntary EMG activity at rest as well as a delayed

relaxation time following voluntary contraction of the vastus medialis and vastus lateralis muscles. The detection of EMG activity during stance and it's sensitivity to load change and to intra-articular anaesthesia was also reported. This study suggests that heightened sensitivity of knee joint proprioceptors contribute to the observed muscular changes.

Peat, Woodbury and Ferkul (150) evaluated 15 subjects with knee joint pathology both pre and post-operatively to examine the activity of four major lower extremity muscle groups in locomotion. EMG data were obtained from surface electrodes placed on the pre-tibial, triceps surae, hamstring and quadriceps muscle groups. In the pretibial muscles, the greatest activity occured at the the stance to swing phase transition. This appeared to serve as a compensatory mechanism for decreased knee movement. In addition, the triceps surae and hamstring muscles demonstrated continuous activity throughout the stance phase as did the quadriceps activity. This activity also appeared to be compensatory. Following total knee replacement, the EMG patterns were found to normalise, possibly confirming the association of muscle dysfunction with the diseased knee joint. Beals et al (87), noted no evidence of clinically significant myopathy or neuropathy in subject with OA of the knee joint studied by electromyography, but found mild fibrillation of the distal

muscles of the foot in two subjects.

Gait analysis

Brinkman and Perry (29) studying the rate and range of knee motion during ambulation in healthy and arthritic subjects found that in comparison with healthy subjects ambulating over a comparable gait-velocity range, arthritic subjects demonstrated reduced rates and range of motion in most instances.

Gyory and associates (146) also found gait velocity and range of knee motion to be lower in arthritic subjects than in healthy subjects. In their study, range of motion was found to correlate with pain, instability of the knee, joint effusion and synovial thickening or bony hypertrophy and standing knee flexion and sagittal knee joint motion was found to be a good predictor of disability.

Wosk and Volishin (152) studied the attenuation of shock waves invading the human locomotion system during gait using accelerometers to evaluate the attenuational capacity of the healthy locomotor system. Results indicated that normal "healthy" individuals may be differentiated into two groups, one presenting with uniform and balanced results and the second with severe imbalance and nonuniformity of results. This investigative method providing an example of a simple diagnostic tool for revealing early deficiencies of the subjects locomotor system.

Stauffer and associates (31), found that the gait mechanics of patients with diseased knee joints showed reduced vertical components of floor reaction force (this is considered an indirect, but proportional approximation of the axial compression force across the knee joint). This finding is substantiated by Grote (201). In addition, an attempt was made to "smooth out" the vertical accelerations during the stance phase. This was accomplished by substituting a more lateral thrust for vertical thrust and by walking at a slower velocity.

Lehman and co-workers (301), however, report that patients with a knee flexion contracture due to degenerative joint disease may demonstrate an increase in the total knee flexion moment when compared to a normal subject with respect to the midstance phase of gait, although a general reduction of vertical floor reaction components as reported by Stauffer et al (31), may be noted when comparing all phases of the gait cycle.

E. ELECTROMYOGRAPHY

The structural unit of contraction is the muscle cell or muscle fibre (158). Groups of muscle fibres are supplied by the terminal branches of a single nerve fibre or axon whose cell body is located in the anterior horn cell of the spinal cord (157,158). The term motor unit is used to describe the single smallest controllable muscular

unit (157,162).

The region of contact between the axon and the muscle fiber is termed the neuromuscular junction or motor end-plate (157). The arrival of a impulse at the neuromuscular junction leads to the release of acetylcholine from the nerve terminal which produces a change in the permeabiity of the post-synaptic membrane (sarcolemma). This gives rise to an ion flux which reverses the resting membrane potential inducing a depolarisation of the corresponding (128,129). The depolarisation (duration approximately .5 msec.) spreads towards both ends of the muscle fibre with a velocity of several m/sec. This travelling electrical activity is referred to as a motor unit action potential (MUAP) and an electromagnetic field in the vicinity of the muscle fibres is generated as a result (155,158,159).

A recording device placed in this field will detect the potential or voltage of this activity (with respect to the ground of the EMG system). The detected waveform will consist of the spatio-temporal summation of individual muscle fibres action potentials originating from muscle fibres in the vicinity of a given electrode or elecrode pairs. The term myoelectric signal is applied to the total signal found at an electrode or differentially between two electrodes and the interference pattern is known as the EMG (155,157,158,159) The depolarisation wave produces

chemical changes within the muscle fibres which initiate a mechanical contraction. The maximum of the force twitch is not reached for some 50 to 100 msec. after the peak of the MUAP, and the decline of the muscle force is slower than that of the electrical activity (157).

The use of EMG as a tool for determining the kinesiological characteristics of muscle function is well documented (153,154).

Nevertheless EMG provides an objective means of quantifying the associated levels of electrical activity during a given motion or task. In particular EMG has been used as an important tool in the clinical and kinesiological evaluation of the knee joint (157,166,191,192).

Surface Electrodes

Where application of wire or needle electrodes is not necessary, surface electrodes can be used in pairs for studying superficial musculature from which a gross representation of activity is desired (33,61)

EMG signals picked up at the surface of the skin represent the potential difference of a given instant between two electrode sites. The potential at any one point will be the resultant of many unit spikes arriving from different sources and having suffered different degrees of attenuation en route (155). While large amounts

of subcutaneous fat may preclude their use, a primary advantage is that they can be easily applied in a standardized manner with virtually no discomfort. Additionally, although, adjacent muscles may contribute to the signal picked up by the surface electrode, it is still possible to provide a suggestion of a given movement pattern or an increase in EMG discharge as long as one does not move the electrodes or change connections when going from one test to another (33,154).

In comparing intramuscular and surface EMG, Bouisset and Maton (163) demonstrate that the activity of a muscle fibre near the surface of a muscle belly represents the activity of all the fibres involved and that in constant velocity isotonic contractions, readings from the surface electrodes are linearly related to those of fine wire intramuscular electrodes.

Komi and Buskirk (164) studied the reproducibility of EMG measures with inserted wire electrodes and surface electrodes and reported high test re-test reliability coefficients (r=.93) for inter-day comparison tests when using surface electrodes.

Soderberg (153) stated that Vittasalo and Komi, in applying surface electrodes over the motor point of the rectus femoris muscle, derived reliability coefficients for maximal contractions to lie between .88 and .91 within the same test day, while Yang and Winter were reported to have

obtained coefficients in the range of .52 to .81 for maximal contractions.

Placement

Surface electrodes are frequently located over identified motor points of muscle for consistant positioning. However, since this location does not always necessarily yield maximum EMG signal values, as demonstrated by Basmajian (162), electrodes may be placed over more suitable anatomically determined landmarks.

Zuniga, Truong and Simons (165) noted that maximum EMG potentials were recorded when the electrodes were placed as close as possible to the middle of the muscle belly.

Kramer and Kuchler (166) in two studies which compared central and peripheral surface electrode location on the biceps and triceps, confirmed that the greatest level of electrical activity was obtained at the middle of the muscle.

Vigreux, Cnockaert and Peruzon (165) studied the factors influencing quantified surface EMG with respect to the biceps muscle and noted that in addition to the influence of electrode with respect to the muscle centre, orientation of the electrode longitudinally along the axis of the muscle picked up twice as much activity as the tranverse deployment.

With regard to the knee joint musculature, Haffajee and co-workers (166), noted that position of electrodes placed along the longitudinal axis of the muscles did not influence the electromyographic recordings markedly.

Before choosing electrode sites, the subject may perform a resisted muscular contraction of the muscle to be studied (167). The EMG operator can then judge the area of greatest muscle bulk and select two sites in the middle of the muscle. The third (ground) electrode can be placed over a bony prominence or at a point equidistant from the two active electrodes (167).

Delagi et al (202) provide a guide for placement of EMG electrodes on a variety of muscles. Attempts to standardise electrode placement also include considerations of inter-electrode distance, electrode size, and electrode material (157,167). Decreasing the interelectrode distance and the use of small electrode sensors will incease the localisation of the signal (157).

Skin Preparation

To ensure that the EMG signals are transmitted with minimal posible loss or distortion, the skin requires special preparation. In addition, the electrodes must possess certain characteristics and a suitable conduction medium should be used between the skin and the electrodes (157).

Normal dry skin has a resistance to the passage of current in the order of hundreds and thousands of Ohms. Skin resistance may be decreased by:

i) Washing the skin with soap and water

ii) Shaving the area

iii) Wiping the area with alcohol

iv) Abrading the electrode sites with fine grade emery paper (157).

Skin resistance may be measured with a standard multimeter and should not exceed 5000 Ohms (157,167).

To ensure that the electrical insulation between the muscle and electrode is kept to a minimum, adhesive strips are normally employed for securing the electrodes and electrical contact is further improved by the use of saline "electrode gel" (157).

Several electrode types are available. In particular, disposable electrodes are readily obtainable and can be applied to the skin after very little training and with reasonable success (162). Generally the metallic parts silver-silver chloride or stainless steel and are set in a plastic base.

Amplification of the EMG signal

Regardless of the type of electrode used, the amplitude of the EMG signal is such that it must be amplified before being displayed or recorded (167). Further

processing of the raw signal is normally achieved by the use of electronic circuits which integrate or filter the raw signal (168). High and low frequency filters change the range or bandwidth of the incoming signals. The frequency of muscle signals recorded by surface electrodes lie in the range of 1-3000 Hz. (169). Grossman and Weiner (170) found muscle signals to range in frequency from zero to ten Hz., with the higher range being more predominant when measured with needle electrodes as compared to surface electrodes. The amplifier must be capable of amplifying signals within the appropiate frequency range and must amplify the signal in a linear manner to eliminate signal distortion (157).

Processing of the EMG signal

The electromyographic signal may be processed in several ways following amplification. One common form of direct processing is the mean voltage estimation (MVE) (167). MVE involves rectification of the signal before it is put through a low pass filter and an envelope of the original signal is produced giving a continuous approximation of the average electrical energy represented in the raw EMG record. Rectification of the signal is normally followed by some form of smoothing to produce a linear envelope,and average EMG activity derived from a composite of signal variables including amplitude, frequency and spike shape is recorded in units of mv-secs.

(169). Inman (169) and co-workers and Zuniga and Simons (172), have shown the mean voltage mode to be proportional to the force produced in the muscle, although the exact nature of this relationship is unclear. Close, Nickel and Todd (144) studying the relationship between motor-unit action potential counts and isometric and isotonic contractions, noted this relationship to be linear with respect to the human soleus muscle, while Zuniga,Truong and Simons (165) found a parabolic relationship between isometric muscle tension and averaged EMG activity with respect to the human biceps muscle. These contradictory findings reflect the dependence of EMG/force relationships upon both experimental setup and the physiological properties of the muscles examined.

Normalization of EMG signals

To eliminate the variation induced by the electrode site and allow data from different muscles and even from different subjects to be accurately compared, the EMG associated with a maximum voluntary effort or the highest EMG value has been selected as a normalizing factor (153). Using this technique, Zuniga et al (165), noted that while the absolute values of the electromyogram differed between electrodes of different sites, the discrepencies were largely cancelled when the average voltage for each electrode site was expressed as a percentage of its maximum

values. Zuniga and Simons (172) credited their ability to discern a parabolic relationship between electromyographic activity and tension through the full range of muscular effort to the minimisation of intersubject variability from normalization of their data. Brownstein et al (257), reported the optimal angle of knee flexion for normal purposes in a female population was 70 degrees. However, others (269) employed angles from 40 to 60 degrees flexion. Normalized EMG is not however an absolute value i.e. mv. and is interpreted as a proportion or a relative value with respect to the MVC condition (292).

EMG and muscle-force relationships

Measuring the force provided by individual muscles in the intact body is problematic (154) and the force generated by an individual muscle may at best be indirectly derived from the torque around the joint upon which several muscles may act synergistically.

Since the force exerted by muscle during contraction depends directly on the excitation which is applied to it, surface electromyographic activity may provide an indirect measurement of the force or tension contributed by a given muscle (154).

The quantitative relationship between the electromyogram and force has been described as linear (169), curvlinear, quadratic or parabolic (172). However,

when the intervals of force are relatively small, this relationship is linear (159).

Under these conditions, it is possible to consider that for any articular position, the torque developed by a muscle is proportional to its integrated rectified EMG (169).

Control of Muscular Force

The natural response of skeletal muscle to force is graduated in terms of motor unit activity and the level of muscular activation may be increased both by recruitment of additional motor units known as spatial recruitment or by an increase in the discharge frequency of the active motor units known as temporal recruitment (159).

Recruitment of motor units is found to be organised according to size, whereby the smaller units are recruited before the larger (174). At low loads, the motor units containing the slow-twitch aerobic fibres are recruited. As tension is increased, the fast-twitch aerobic fibres are recruited (175).

Stein et al (176), note that the largest contribution to motor unit recruitment occurs at low force levels, while the contribution of the increased firing rate becomes more important at higher force levels, generally in excess of 75 percent of maximum isometric strength (189).

A third mechanism related to the graduation of

contraction is synchronisation between the motor units which are firing. This is most often associated with fatigue (159,176).

EMG AND MUSCLE MECHANICS

The mechanical properties of the contractile system are expressed in the relationship between force and length and force and velocity.

Length-tension relationship

According to the length-tension relationship described by Blix (178), the force exerted by a muscle in an isometric contraction depends upon it's length. These changes in the force lever arm are more or less compensated for by changes in the level of muscular excitation. In general this relationship is such that the integrated EMG magnitude for any given force is greater for a shorter muscle length and is diminished at a longer muscle length.

The force-length function is also noted to differ with respect to the direction of the muscle fibres (179) or to the ratio of type I/type II fibres within the muscle (180).

Influence of velocity

A reciprical relationship between the velocity of contraction of isolated muscle fibres and tension has been

demonstrated by Hill (181).

Bigland and Lippold (182) noted the interdependence between tension, velocity of contraction and the integrated EMG and indicated that the slope representing the relationship between the integrated EMG and tension was greater when the muscle shortened at constant velocity than when it lengthened. Komi (183) found the relationship between muscle tension, EMG activity and velocity of contraction under concentric and eccentric conditions to be similar regardless of the type of contraction used or the velocity of contraction selected.

In contrast, Nelson and associates (184), reported contradictory results for the contraction velocities of the tibialis anterior and soleus muscles. For instance, when contraction velocities were increased from 24 degrees/sec to 2[illegible] degrees/sec, the integrated EMG decreased 81.4 percent for the tibialis anterior muscle and 86 percent for the soleus muscle. This finding is substantiated by Barnes (185) who found that motor-unit activity decreased as the contractile velocity increased.

Electromechanical Delay

In contraction of skeletal muscle a delay exists between the onset of electrical activity and the development of muscle tension (186). This delay known as electromechanical delay (EMD) was formulated by Hill (187)

who postulated a two component model of muscle in which the slow development of tension was due to the presence of elastic elements aligned in series with the contractile element. Other experiments utilising concentric and eccentric contractions and different muscle groups have led to the assumption that EMD is dependent upon the structural components of skeletal muscle (186,187).

In a study conducted by Komi and Viitasalo (187), the relationship between EMD and muscle fibre composition was assessed under voluntary and reflex contractions. These authors report that while no relationship could be established for the EMD measured under reflex conditions, the EMD under voluntary contraction was related to muscle fibre composition and was shorter in subjects with a high percentage of fast twitch fibres of the vastus lateralis muscle.

A study of the electro-mechanical response time and rate of force development described by Bell and Jacobs (189) indicates a shorter response time for males than for females. This difference is speculated to be due to structural differences in tissue elasticity and to the proportion of fast twitch fibers in the contracting musculature. It is suggested furthermore, that prior activity history and more probably daily activities are likely to elicit a strength training effect in favour of the male both in terms of muscle stiffness and in the

proportion of fast twitch fibres in given muscle.

ELECTROMYOGRAPHY OF THE KNEE JOINT EXTENSORS

The nature of interplay amongst the four heads of the quadriceps femoris muscle (QF) has been studied by several workers (190,191,192).

As documented by Basmajian, Harden and Reginis (191) the interplay of activity between the vasti and the rectus femoris (RF) muscle in normal subjects during leg extension and squatting motions, demonstrates notable variation between the different heads of the quadriceps femoris muscle as well as considerable standard deviations arising from individual differences. In addition, the late onset and early cessation of activity in the rectus femoris muscle during the squatting was noted.

Similarly Ravaglia (192) noted the asynchronous activity of the vasti and RF in rising from a sitting to a standing posture and stated in addition, that RF was the most important muscle leading the knee into extension. Wheatley and Jhanke (193) however, found the RF muscle inititaed extension while the vasti showed greater activity at the end of movement.

While Le Sage and Le Bar (cited in 157) considered the vastus medialis (VM) and vastus lateralis (VL) to act as initiators of knee extension, Pocock (195) concluded that there was little variation in temporal sequence

amongst these these three QF muscle components and that the EMG patterns were similar throughout the entire range of knee extension.

Similarly Adler and co-workers (196) concluded that during the gait cycle, the QF muscles acted as a unit to produce knee extension.

The difference in outcomes of these studies and others not documented here, complicate definitive statements about the timing of muscle action and the tentative role of individual muscle components in any given activity. These differences may reflect the various methodologies used and instrumentation employed, as well as individual variations amongst other factors. In addition, the quadriceps contraction force depends partly on the degree of motor activity and partly on muscle length relative to the resting length (166) and thus activation patterns in the sitting position are likely to differ from those in the standing position. In addition, changes in the hip joint angle are likely to influence the length of the rectus femoris thereby altering its activity.

QF muscle response also appears to be influenced by certain intrinsic characteristics of the knee joint that tend to change the moment arms of the muscles as the knee moves. Andriacchi and associates (199) note in particular that QF muscle activity is reduced in the presence of a flexion abduction moment, while activity is notably

increased in the presence of a flexion-adduction moment.

With regard to knee joint angle and mechanical influences, Murphey, Blanton and Biggs (200) noted increased QF muscle activity to occur during stance in subjects who habitually flex their knees and diminished activity in subjects showing hyperextension of the knee joints in this position.

F: TESTING WITH AN ISOKINETIC DYNAMOMETER

The concept of isokinetic exercise was developed during the early 1960's by J.J Perrine and first reported by Hislop and Perrine (203). An isokinetic contraction is a dynamic muscle activity performed at constant angular velocity. The required speed of motion is generally selected and controlled using an isokinetic dynamometer, such as the Cybex II. Normally if the force of dynamic muscle contraction is unchecked, this results in acceleration. However, the Cybex dynamometor provides resistance to movement when the exercising limb attempts to exceed the preselected speed (204). The machine is thus specifically designed to maintain limb movement at a constant angular velocity and provides for accommodating resistance (203). The muscular force applied to the lever arm is sensed by an internal load cell and transmitted to a recording device to produce a torque curve (206).

Isokinetic muscle testing may provide an objective measure of muscular performance with respect to torque, as well as timing related to torque amongst other factors.

Richards (207) commented that since the resistance given by the dynamometer never exceeds the force applied, very weak patients can be tested without the risk of overloading the muscles participating in the movement.

Reliability and Validity of the Cybex II

The reliability with respect to the measurement of muscle strength and angular velocity are reported to be high (78).

Richards (78) states that Moffroid (208) and associates showed that torque values measured by the Cybex dynamometer increase linearly in response to increasing weight placed on the lever arm at the same distance from the axis of rotation and at the same angular position, with a test re-test reliability coefficient of r=.995.

It was also reported in addition, that Moffroid (208) found the coefficient of validity for torque measures at various angles to be r=.999. The validity of measurements of the area under the torque curve i.e. work measures were also established (208).

Measures from the torque curve

Many characteristics of muscular performance can be

measured from the recording of torque curves and limb position. Peak torque, defined as the highest point on the generated torque curve, is normally recorded in foot pounds or Newton metres (204). Depending on the test protocol, this value reported in the literature, may represent the highest single value obained during a series of repetitions (207) or the mean value of a series of repetitions (209). If the purpose of the test is to determine an individuals maximum capability to generate torque, the highest single value may be an appropiate indicator. In assessment of the injured or deconditioned individual however, either the mean or the standard deviation or the range of values over a series of repetitions may be a better measure of functional capability (214).

Time factors measured in relation to the torque curve include time to peak torque, time rate of tension development, time peak torque is held and force time decrement (205). Watkins et al (214), used time characteristics of isokinetic contractions to describe knee extensor and flexor muscle activity in patients with hemiparesis, while Murray and associates (212), examined time to peak torque in normal individuals during isometric knee flexion and extension contractions. No similar studies have been reported with regard to the musculature of the osteoarthritic knee joint.

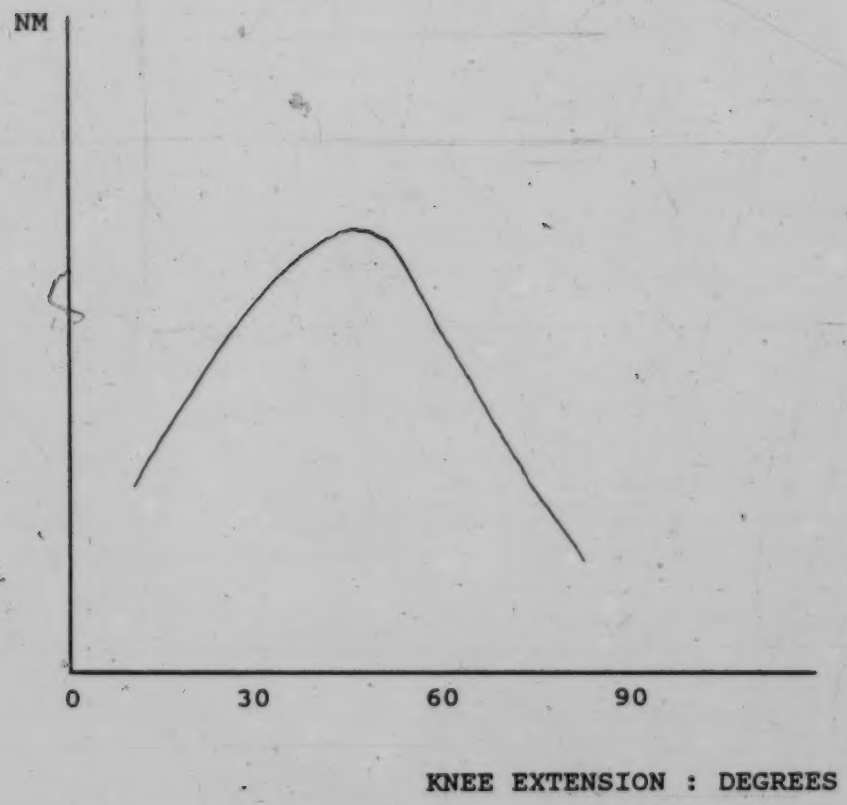

Figure 2.1. Knee extension strength curve. Adapted from (166,212,216)

The Strength Curve

The shape of the torque-angle (strength) curve is determined by two primary factors: (i) the length-tension effect and (2) the perpendicular distance between the line of pull of the muscle and the axis of the joint which the muscle acts. In the knee joint this is found to vary at different joint angles (203). In addition, the orientation of the muscle fibres with respect to the lines of action of individual muscles may also influence the torque output. In comparison to the hamstring musculature where the muscle fibres are aligned parallel to the line of action of the muscle, large portions of the knee extensor muscles, particularly of the vastus medialis and vastus lateralis muscles are aligned obliquely to the action line of the QF muscle. Here the component of tension pulling proximally on the patellar ligament is proportional to the cosine of the angle subtended between the muscle fibres and the total action line (215).

The force-angle relationship during an isometric contraction may produce a strength curve as depicted in figure 2.1. Through the range of movement depicted, the length effect acts to increase muscle tension from the shortest to the longer muscle lengths. Here the optimal angle of pull may lie between 30 and 60 degrees with respect to the knee joint (166,212,215).

Force velocity curves

The classic in vitro force velocity relationship described by Fenn and Marsh (217) and Hill (218) was hyperbolic in nature. This curve reflected a specific inverse relationship between work capacity and speed. While Thorstensson et al (219) noted curves which closely resembled the force velocity relationship of isolated muscle with respect to the knee joint extensors, Perrine and Edgerton (220) described marked differences between the in vitro force-velocity relationship and the in vivo torque -velocity relationship described above. Several workers have noted that torque output decreases as the velocity of contraction increases (185,219,220,221).

Normal knee velocity is found to range from approximately 50 degrees per sec in the stance phase of gait to 90 degrees per sec in the swing phase (222) and the forces produced at or above 96 degrees per sec are found related to the fast twitch fibre component (86).

H: PHYSICAL THERAPY TREATMENT APPROACHES IN THE MANAGEMENT OF OSTEOARTHRITIS

Lawrence and associates (223), assessing the value of physical treatment for rheumatic disease, suggested the problem be approached from two different perspectives:

1) whether the remedy relieves the pain and
2) whether it hastens recovery or makes it more complete.

With regard to the management of pain, protection from excess weight-bearing (97,224), rest (3), heat treatments such as hot packs (151), shortwave diathermy (228,229), microwave (227), infra-red (151), wax baths and ultra-sound (151) as well as interferential current (240), massage (151), cycloidal vibration (230), trancutaneous electrical stimulation (231), traction (242) and ice therapy (226) have been described as efficacious. However few well controlled clinical studies have been published to evaluate any of these approaches.

Although decreases in muscle strength and endurance (30) play a significant role in the degree of functional impairment associated with OA, and selective changes in size and distribution of fiber type in the vastus muscles have been found in relation to gonarthrosis (232), relatively little has been published regarding the value of exercise in the treatment of OA and with the exception of Janda (24) no comprehensive approach has been formulated.

However, several workers have postulated the potential value of exercise in this regard. Hamilton and co-workers (236) on the basis of their general clinical impressions cited that exercise was potentially a most useful measure and McCloy (97) suggested that in terms of exercise, selective muscle strengthening was an important element. Doyle (3) emphasised exercise tailored to the individual and felt muscle power should be improved to

support damaged joints. Vloebergs (237), in acknowledging the role of muscle hypotonia in OA, proposed certain principles of treatment with emphasis on the use of appropiate exercise in the management of gonarthrosis. In contrast Clarke and associates (226) stated that while active treatment of the quadriceps may prevent wasting in acute states, in a more chronic state muscle wasting can be prevented by encouraging general activity.

In evaluating the role of exercise therapy in the treatment of the osteoarthritic knee joint, Quirk et al (240), employed an exercise regime consisting of straight leg raise, performed twice daily, with weights added at 1 lb. increments according to the subjects ability. In addition, free active flexion exercises were performed at each exercise session. While three subjects were found to deteriorate as a result of the exercise programme, a clinically significant improvement in the overall group of subjects was noted.

Adler (233) used inner range quadriceps exercises as well as squatting, in devising a self-care management for persons with gonarthrosis and found 53 of 72 affected joints to improve functionally although the criterion measures were not specified.

Chamberlain and associates (229), using a similar approach noted improvment in the majority of their subject with respect to range of motion, function, pain score,

strength and endurance.

Luckhurst and associates (239) noted the effect of isometric quadriceps exercise in subjects with RA and OA of the knee joint. Results reported on the five osteoarthritic subjects in this study showed that despite adequate strength gains, this approach did not improve the functional ability of these subjects to a measurable extent. Although this finding was attributed to the degree of prevailing joint pathology affecting joint mechanics and the presence of pain and although muscle atrophy was noted, the quadriceps muscle was considered by these workers to be normal. In light of more recent findings (83,84) this statement appears to be short sighted.

The effect of exercise on cartilage structure and function has been documented (22,234,235). In addition, more recent studies indicate the probability of cartilage regeneration under optimal physical condition (246,248) Few attempts have been made however, to apply these fundamental observations to improving the status of the joint in its degenerative state and in particular, the rational for and application of exercise for osteoarthritis remains largely empirical. The paucity of study in this regard, together with the observation that muscle is significantly altered (36) or implicated (82) in OA, indicates the need for improved understanding of muscle function in this disease if rational and effective

treatment is sought.

III. METHODS

A. SUBJECTS

Thirty subjects in the age range of 18 to 65 were entered into the study. The subjects were assigned to one of three groups on the basis of knee joint status as follows:

1. Normal
2. At risk
3. osteoarthritic.

Inclusion Criteria.

Normal Group:

1. Healthy females presenting with normal knee joint function and alignment.

At risk Group:

1. Healthy females presenting with clinical evidence of tibial torsion and/or varus malalignment of the lower limb(s) with or without concomitant clinical signs of chondromalacia patellae.

OA Group:

1. Knee pain plus three of six signs as follows:
   a. age > 50 years.
   b. stiffness < than 30 minutes.
   c. crepitus

d. bony tenderness

e. bony enlargement

f. no palpable warmth

All patients had radiographic evidence of gonarthrosis.

Subjects meeting any of the following criteria were excluded from the study:

1. history of any hip, knee, or ankle surgery, or major knee joint injury.
2. presence of musculoskeletal disease other than idiopathic OA of the knee joint
3. history of cardiovascular disease such as angina or any cardiac ailment for which subjects were receiving treatment
4. history of primary neuromuscular disease
5. unable to follow instructions
6. persons engaging in elite athletic activity
7. persons unable to walk a distance of 250 m. with or without aids.
8. previous training on an isokinetic device.

B. RESEARCH DESIGN

Examinations of the knee joint in healthy females, age range 18 to 29 years, were carried out during the period December, 1987 to March, 1988 in the Department of Medicine, University of Alberta Hospital by a

Figure 3.1. FLOW DIAGRAM OF STUDY PROTOCOL

Identification of subjects

Examined for eligibility NO Excluded from Study

YES

Assigned to one of three study groups

Informed consent form read and signed

Testing Protocol

Familiarisation and clinical measurement

Electrode application

Stabilisation of subject in appropiate test position

Practice session; two submaximal; one maximal effort

Rest period : five minutes

Test : three MVC with two min.interval between

Rest period: five minutes

Repeat practice and test sessions in all designated positions.

Rheumatologist. In addition, female subjects, age range 43 to 69 years, diagnosed with OA of one or both knee joints were referred to and recruited by the investigator provided they were willing to partake in the test session and fulfilled the criteria for idiopathic OA of one or both knee joints (Appendix A). Having met the inclusion criteria, the subjects were assigned to one of three groups previously described. All subjects read, signed and received a copy of an informed consent document (Appendix E). Consenting subjects then attended a single test session during which time measures of electromyographic activity and torque were recorded during seated knee extension with respect to the quadriceps femoris muscle group. No specific practice session was held, but all subjects were given adequate time and opportunity to aquaint themselves with the test apparatus and to perform a standardised series of pre-test contractions. A flow diagram of the study protocol and test procedures is presented in figure 3.1.

C: EVALUATIVE PROTOCOL

CLINICAL

Subjects were assessed in a standardised manner with reference to knee joint stability, alignment, effusion and crepitus amongst other factors. Demographic data including height (in metres) and weight (in kilograms) were also recorded. An evaluation form was completed for all subjects

(Appendix C). Prior to testing, clinical evidence of tibial torsion was confirmed by placing the leg in a free-hanging resting posture and noting the position assumed by the foot with respect to the frontal plane of the body. Alignment was considered normal if the foot remained in a neutral position with respect to the tibia. Clinical evidence of varus was confirmed in both lying and standing. In supine, a tape was stretched taut and centred at the upper end of the thigh over the middle of the femoral head (halfway between the anterosuperior spine of the pelvis and the pubic tubercle) and over the middle of the patella. If the lower end of the tape lay over the midline of the ankle, alignment was considered normal. If the ankle lay medially, the knee tibia was said to be in a varus position (290) In standing, subjects were viewed from the anterior aspect when the patellae faced forwards and the medial aspects of the knees and medial malleoli of both limbs were as close together as possible. If the ankles touched and the knees did not, the subject was said to have genu varum (291). Measurement (in cm) were also made of thigh length (anterosuperior spine to medial joint line knee), shank length (medial joint line of knee to medial malleolus) and thigh circumference (10cm. above superior aspect of the patella) using a standard tape measure with the subject positioned in supine.

FUNCTION

Osteoarthritic subjects answered a questionnaire concerning their functional abilities and their pain patterns (Appendix D). These evaluations were not carried out on healthy subjects.

EMG MEASURES

Full wave rectified and linear envelope detected EMG patterns were recorded from three pairs of electrodes placed upon representative sites of the QF muscle. The peak, and integrated EMG values were measured manually. Measures obtained were normalized against the best measure read at 60 degrees knee flexion. This value was used in the statistical analysis.

TORQUE MEASURES

Peak torque and time to peak torque (TTPT) records were determined from the knee extension torque curves. The initial slope of the torque curve was also calculated and used in the statistical analysis.

D:TESTING

Evaluative procedures were carried out in the Department of Physical Therapy, University of Alberta. Prior to the study both the Leaf Electronics System VII EMG

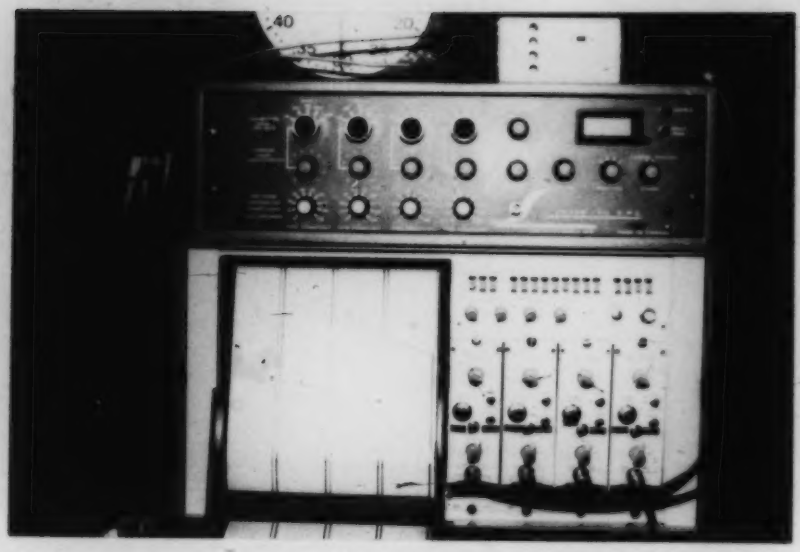

PLATE 3.1. EMG Amplifier and remote transponder unit.

Plate 3.2. Isokinetic dynamometor and EMG amplifier

Amplifiers[1] and the Cybex II isokinetic dynamometer[2] used for testing purposes had been calibrated according to manufacturers specifications (Appendix E,H). Additionally, the dynamometor was calibrated with known weights both during isometric as well as isokinetic contractions prior to the testing of each subject. Baseline settings and battery checks were carried out prior to each test to ensure standardisation of EMG and torque measures. Optimal amplifier gain settings, smoothing time constants and frequency cut off for each channel were determined during a pilot study and were kept constant for all subjects. In particular, amplifier gain settings were chosen to prevent clipping but to ensure adequate amplification of myoelectric activity.

Plates 3.1 an 3.2 display the testing apparatus and the position of the subject relative to it.

The specifications of the Leaf VII System Amplifier are presented in Appendix F. This device facilitated detection of electrical activity from selected points of the QF muscle of a single knee joint for each subject. Torque production during reference and test movements was made possible by the use of the Cybex II isokinetic dynamometer.

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[1]Leaf Electronics System VII 4-Channel Electromyograph and Amplifier; Leaf Electronics Ltd., Suite 102, 11804 - 124 Street, Edmonton, Alberta, T5L 0M3.

[2]Cybex. A division of Lumex Inc. 2100 Smithtown Avenue. Ronkonkoma, N.Y. 11779

Three channels of the Leaf Electronic Amplification System were connected to the bioelectric amplifiers of a Hewlett Packard 7754A[3] recorder via cables attached individually to each of three integrated output mode channels on the Leaf Amplifier. The full wave rectified and amplified signals for each channel were then recorded on thermal paper by a pen recorder.

The integrater smoothing time constant was set at 200ms. This value was derived from observations made during the pilot study and was based on the same value employed by Richards et al (273) in a study of the rheumatoid knee joint. The artefact suppression was set at 60 Hz. The EMG amplification settings were adjusted independently and the gain for channels one, two and three were set at 10mv, 50mv and 20mv per 10mm divisions of graph paper. The upper cutoff frequency selected was 10Hz. The chart speed on the four channel Hewlett Packard Recorder was set at 10 mm/s for both isometric and isokinetic test movements.

Leads for the surface electrodes were connected to the remote transponder unit of the Leaf System VII Electromyograph and the selective band filter on the unit was used to filter potential 60 Hz interference from the atmosphere. The EMG signal was transmitted from the transponder unit through an input cable to the main EMG

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[3]Hewlett-Packard 4-Channel Thermal Tip Recorder; Hewlett-Packard (Canada) Ltd., 1162A - 168 Street, Edmonton, Alberta, T5M 3T9.

unit, and thereafter to the Hewlett Packard Recorder.

In order to synchronise the torque recordings with the EMG registrations the fourth channel of the Hewlett Packard recorder was used.

The placement of the surface electrodes was done as follows:- The subject lay in supine and appropiate sites of the vastus medialis and lateralis muscles as well as the rectus femoris were located by inspection, measurement of landmarks described by Delagi and associates (202) and by palpation of the muscle belly on active contraction. These sites were demarcated and the skin prepared for testing by rubbing the area briskly with a paper towel soaked in isopropyl alcohol. Disposable electrode pairs HP 1444 5C[4] were then applied to the skin surface over the chosen sites of the three muscle bellies. These were placed along the long axis of the muscles with an interelectrode distance of 2 centimetres measured between their centres. The single ground electrode was placed on the lateral prominance of the tibia. Standard adhesive tape was used to secure the electrode wires to minimize movement artifact.

Following electrode placement the subjects were seated in the exercise chair with the thigh supported by towelling to provide a horizontal orientation of the thigh when the knee was extended. This approach was used by

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[4]Hewlett Packard. Medical Products Group. Waltham, Mass. 02154.

Plate 3.3. Subject positioned for isokinetic contraction

Plate 3.4. Subject positioned for isometric contraction at 60 degrees flexion

Henley (292) in a similar knee joint study. Stabilisation was provided by a 10 cm. wide strap centred across the pelvis and anchored through two openings in the back rest. A thigh stabilisation strap, attached to the seat of the table, was applied to the lower thigh region of the leg tested and centered approximately 10cm. above the patella. Due to variations in femoral lengths, additional backrest inserts were used to maintain an appropiate distance between the edge of the table and the limb. The back support was adjusted such that the hip joint angle was approximately 105 degrees with respect to thigh. Deutsch (213) found that if total quadriceps muscle activity was of concern, knee extension with hip flexion in the range of 90 to 135 would be appropiate.

The axis of rotation of the input shaft of the dynamometer was aligned with the anatomical axis of the subjects knee i.e. placed alongside the lateral joint line of the knee when the knee was resting in approximately 90 degrees of flexion. The lever arm was adjusted and attached to the lower limb at a point of comfort above the malleoli with a padded strap. To ensure comfort and appropiate alignment during movement, the knee was passively extended once through the range of motion from knee flexion to knee extension (209,241).

For testing the knee was placed randomly in one of three starting positions, namely, the angles of 30, 60 or

Plate 3.5. Knee positioned at 60 degrees flexion

PAGINATION ERROR

PLATE 3.6 ("PAGE 95")
SHOULD BE ON PAGE 91.

PLEASE, REFER TO LIST
OF PLATES ON PAGE XVI.

ERREUR DANS LA PAGINATION

LA PAGE 91 EST DEVENUE
PAR ERREUR LA PAGE 95.

VOIR LISTE DES PLANCHES
A LA PAGE XVI.

Plate 3.6 Subject positioned for isometric contraction

90 degrees knee flexion measured relative to the horizontal plane which was defined as zero degrees flexion. These angles were read directly from the dynamometor goniometer and verified with a standard manual goniometer placed alongside the lateral joint line of the knee and aligned with the bony landmarks of the greater trochanter of the femur and the lateral malleolus of the fibula. The measurement was taken as the subject contracted isometrically against the arm of the Cybex to allow for the compressibility of soft tissue and give provided by the padded strap and the input shaft occuring during movement (241,292). The dynamometor range limiting device was used to ensure consistancy of measures made at each angle.

Both isometric and isokinetic measures were made. Prior to each test sequence the subject performed two submaximal (50%) contractions and one maximal contraction as prescribed by Mawdsley et al (210). The term submaximal was described as a contractile effort requiring no more than approximately 50% of a subjects maximum capability. For all test sequences, the subjects were asked to maintain a standard upright head-body position. Tests were conducted with the subjects folding their arms across their chests. During isometric tests the subjects were instructed to extend their knees smoothly but maximally and to maintain this effort for a period of three seconds. The verbal command "ready and push" was employed consistently for all

Plate 3.7. Subject positioned for isometric contraction

Plate 3.8. Subject stabilised for isometric contraction

all subjects. An event marker recorded the onset of the contraction period, three seconds duration, was monitored with a stop watch. Verbal commands were given during contraction period to facilitate testing. Three contractile efforts were recorded at both 30 and 60 degrees knee flexion. The readings noted at the 60 degree angle of knee flexion were used as a reference for calculating the parameters of EMG activity employed in the statistical analysis.

Testing also included three trials of isokinetic contraction performed at angular velocity of 90 degrees/s. Each isokinetic test started from an angle of 90 degrees knee flexion and proceeded towards the subjects maximal range of knee extension. The subjects were instructed to "kick as hard and as fast as possible" for each isokinetic trial. The verbal command was "ready and kick". The subjects performed the movement into extension as described and were allowed to return to the starting position at their own pace (78).

All three trials were separated by a five minute rest period, allowing for the apparatus to be adjusted to the next position. A two minute rest interval was given between each contraction period within a particular trial. Normal subjects had their dominant knee tested i.e. the knee favored to kick a ball (207,241), while arthritic subjects had their affected leg or weaker leg tested in the case of

Plate 3.9. Axis of dynamometor aligned with axis of knee joint.

SAMPLE TRACE ISOMETRIC

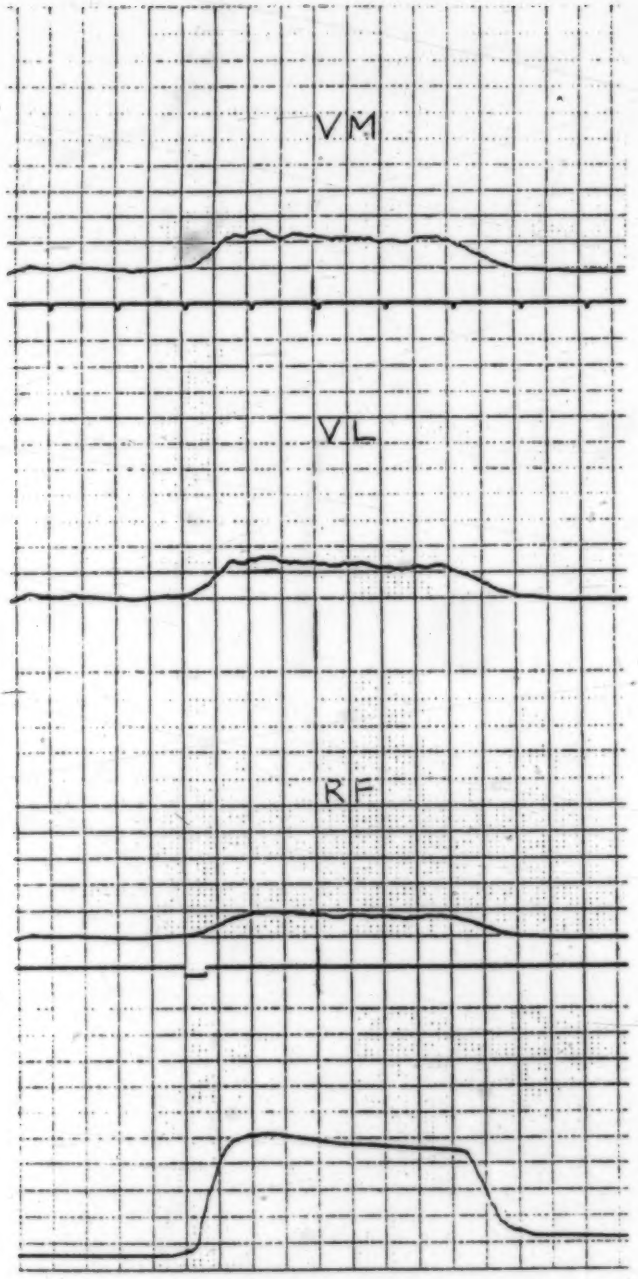

Figure 3.2..

output of effort during each test sequence and the Cybex monitor was used to provide visual feedback as suggested by Figoni and Morris (247).

During all trials, the full wave rectified and linear envelope detected EMG signals were amplified and recorded on thermal paper together with the knee extension torque measures. The best readings over each of three trials were used for analytical purposes. These were defined as readings exhibiting the highest peak torque values on any one of three repetitions of the muscle test.

E: MEASURES

Measures made from the EMG and torque curves included:

a) Peak EMG

b) integrated EMG (IEMG)

d) IEMG\torque ratio

e) initial rate of tension development

f) time rate of tension develpoment-isokinetic

F: DATA ANALYSIS

The dependent variables were the following;

a) EMG, peak and integrated, measured in mm. and normalized against measures at MVC 60 degrees knee

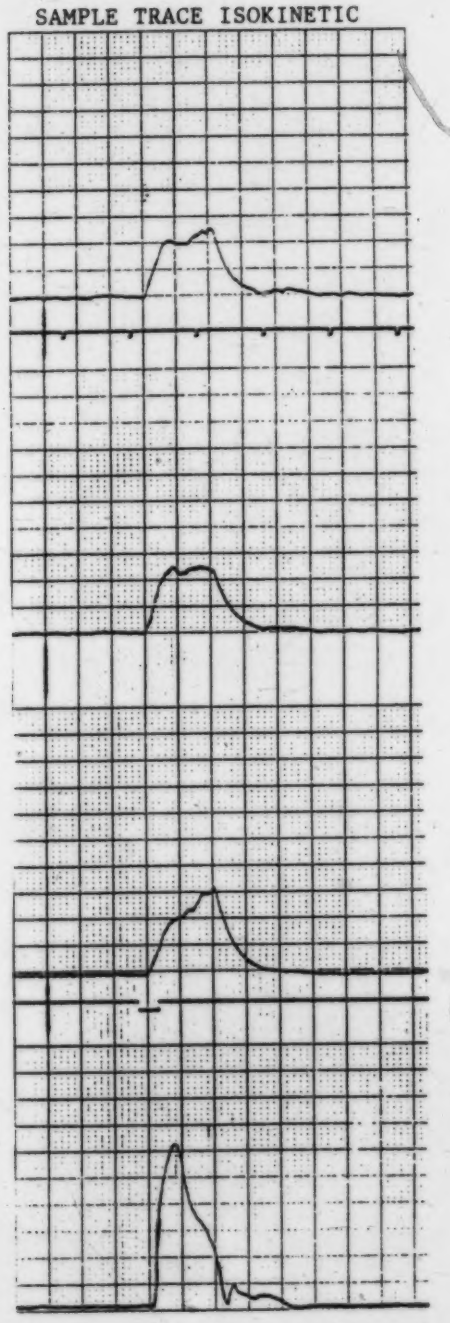

Figure 3.3.

flexion, expressed as a percentage.

b) the rate of tension development measured in Nm/s.

c) electromechanical delay measured in msec.

e) IEMG/force ratio, expressed in percent.

f) torque measures expressed in Nm.

Analysis of variance tests (ANOVA) were employed to determine the significance of these variables. The PC ANOVA[5] statistical package was used. Where appropiate, post-hoc analyses were conducted using the Newman-Keuls test. The minimal level for statistical significance was set at $p < .05$.

G: ETHICAL CONSIDERATIONS

This project received the approval of the Student Projects Ethical Review Committee (SPERC) of the Department of Physical Therapy, as well as approval from the Faculty of Medicine, University of Alberta.

All subjects read and provided informed consent prior to their admittance into the study (Appendix E). All subjects were verbally advised regarding the nature of their participation in the study and were assured they were free to leave the study with no penalty at any time. Patients were also imformed that they were not being offered a treatment for their condition, but that the

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[5] PC ANOVA, 9010 Reseda Boulevard, Suite 222, Northridge, California 91324-3971.

information would be used solely with the intent of deriving improved knowledge concerning muscle function in osteoarthritis.

IV. RESULTS AND DISCUSSION

The investigation of EMG activity and time factors associated with tension development provided both descriptive and quantitative imformation.

These data are presented and discussed in five sections as follows:

1) anthropometric data
2) electromyographic data
3) electromyographic-force ratios
4) time factors related to tension development
5) torque measures.

The overall findings are summarised at the end of the chapter and related to the study objectives.

All statistical measures employed a one-way or two-way analysis of variance (ANOVA) package, and significance was determined at the .05 level of probability. The Newman-Keuls test was used to determine post-hoc differences between means when the F ratio was found significant. Statistically significant values are indicated by an asterisk* throughout the text.

A.SUBJECTS

A total of 27 female subjects with normal and pathological knee joints were included in this study. Due to problems in recording accurately, EMG data from two

osteoarthritic subjects was excluded from the analysis. Subjects in the normal and "risk" groups were students at the University of Alberta recruited on a voluntary basis. In terms of activity level, the majority (60%) of these individuals were sedentary, while the remainder pursued a variety of recreational activities. Osteoarthritic subjects were generally retired persons or persons unable to work as a result of functional disability (66%), while the remaining subjects pursued sedentary occupations in the workforce. Seven subjects had bilateral OA. The duration of diagnosis varied from two to ten years. All subjects were on medication for control of pain and imflammation. Functional ability as determined by questionnaire established that the majority of osteoarthritic subjects were functional at the grade II to grade III level dependant upon disease status which was usually described as varying from severe to mild or moderate. In terms of functional disability, pain appeared to be the most significant factor affecting all subjects and generally varied in intensity from mild pain with fatique to constant severe pain. Although walking distance appeared somewhat limited in the majority of subjects, greater problems were noted with regard to functional activities involving stairs and in rising from a seated to a standing position. In addition, most subjects complained of fatigue following weight bearing activity. The majority (77%) of subjects

presented with clinical signs of knee joint effusion. These observations concur with similar findings reported in the literature (30,32,127).

TABLE 4.1: ANTHROPOMETRIC DATA:

CHARACTERISTIC	NORMAL GROUP n=9	RISK GROUP n=9	OSTEOARTHRITIC GROUP n=9
Age (years)	22.33 (19-28)	24.00 (19-28)	60.33** (47-69)
Height (cms.)	168* (158-174)	162 (151-168.5)	161 (156-165)
Weight (kilos.)	62.12 (46.5-80)	62.28 (53-86.5)	79.11** (60-113)
Weight/Height^2	22.04 (16.4-27.7)	23.97 (18.79-35)	29.71** (23.4-33.09)
Thigh length (cms)	42.00 (40-46)	44.11 (40-48)	43.56 (38-46)
Shank-length (cms)	39.33 (37-42)	38.44 (37-42)	37.44 (35-40)
Thigh circumference (cms)	44.5 (39-58)	47.66 (43-59)	51.27 (41-69)

Data are presented as the mean and range of values

** statistically different from normal and risk

* statistically different from risk and OA subjects

Analysis of the anthropometric data (table 4.1) revealed that with the exception of height which was significantly greater in the normal group (p=.01), no statistically significant differences were found between subjects in the normal and risk groups. Although Richards (78) attributed little significance to height with respect to the outcome of strength measures of the knee joint, the factor of height in the normal group can not be discounted as an influence on test measures studied.

Significant differences between arthritic subjects and the two healthy groups were noted with respect to age (p=.01) and weight (p=.001). In light of the acknowledged association between age and OA (2,5), this finding was expected. However, it should be noted that due to problems in acquiring subjects in the "disease free state" with any degree of certainty in an age matched population, osteoarthritic subjects were compared with younger healthy individuals on the basis of joint alignment. Since varus malalignment and/or tibial torsion are documented (25,26,27) risk factors in OA and since a majority of female osteoarthritics present with similar tibial alignment (249), this approach also provided an opportunity to define both differences and similarities which might be of predictive value in the pathogenesis of OA. Regardless, it should be noted that the effect of age, as well as weight on the results reported in this study remains

unknown with respect to the osteoarthritic subjects as a result.

In reviewing these data, a trend by the risk group to higher height-weight ratio values in the direction of those presented by arthritic subjects was noted. Mean age values also tended to be higher than those of normal subjects. Reduced shank length and increased thigh circumference noted in the "risk" subjects also displayed a trend in favor of arthritic subjects. This apparent tendency towards mesomorphism is of interest in light of the findings by Grote (201), who noted the mesomorphic component of the somatotype in both male and female osteoarthritics to be greater than normal. Solomon (50) also made this observation in an earlier study. Leach (249) concluded that significantly over-weight women over the age of 50 with varus deformity have a high risk of developing OA of the knee. The higher score in mesomorphy in relation to OA is difficult to explain, although Grote (201) suggests the influence of muscular physique as a factor either as a cause or a consequence of the disease. Hartz and associates (250) state that the greater femoral musculature in men provide enhanced stabilisation of the knee joint, absorbing more of the impulse associated with loading, than the same musculature in women. These workers also imply that obesity would be an increased risk factor for OA in the female population as a result.

B. ELECTROMYOGRAPHIC DATA

a) Peak EMG

Peak EMG values, defined as the highest amplitude of activity recorded above the baseline during both isometric and isokinetic conditions, were recorded in mm (intra-rater reliability for manual calculations of these data r=.99; mean test re-test reliability r=.91). Values produced isometrically at an angle of 60 degrees knee flexion were used in the normalising process. That is, all other readings were expressed as a percentage of this value. Mean values for each muscle, as well as group means were calculated and used in the statistical analyses.

Graphic representation of these values are shown in figures 4.1 and 4.2; and tables 4.2 and 4.3 and summarise results for normalised peak EMG values both isometric and isokinetic. Isokinetic values were defined as "fast" to assist in the interpretation of the data.

In this study, peak values were chosen to reflect the point at which coincident spikes might overlap i.e. potentially at the point of maximal muscle tension development and therefore representative of the total EMG pattern. Blanchi and Vila (269) found the amplitude of rectified EMG to be a relevant parameter for the evaluation of muscle function during maximal voluntary contractions.

TABLE 4.2: PEAK ISOMETRIC EMG AT 30 DEGREES

Mean (x) and standard deviations (SD) of normalised scores

		GROUPS		
MUSCLE	STAT	NORMAL	RISK	OSTEOARTHRITIC
VM	$\bar{x}$	110.889	113.889	142.714
	SD	15.42	36.61	61.68
VL	$\bar{x}$	108.889	119.889	129.286
	SD	20.82	33.06	52.95
RF	$\bar{x}$	106.333	121.333	165.000*
	SD	17.13	20.07	42.42
QF	$\bar{x}$	108.704	118.370	145.667*
	SD	17.79	27.71	52.35

*significantly different from risk and normal

Results of ANOVA tests performed on peak EMG values, indicate that during isometric contraction (table 4.2), activity patterns displayed by normal and risk subjects were similar with respect to all muscle groups. However higher mean values were recorded for arthritic subjects,

with respect to all muscle groups. During isokinetic contraction (table 4.3), a tendency to increased peak activity was displayed by all three groups, with the activity of the risk group showing a relatively greater increase in this regard. As a result, the activity pattern displayed by risk subjects during isokinetic contraction closely resembled that produced by arthritic subjects with slight variation, such that the activity of the VL muscle which was higher and the RF activity lower in the risk group.

TABLE 4.3: PEAK EMG ISOKINETIC

Mean ($\bar{x}$) and standard deviations (SD) of normalised scores

		GROUPS		
MUSCLES	**STAT**	**NORMAL**	**RISK**	**OSTEOARTHRITIC**
VM	$\bar{x}$	125.889	164.333	161.714
	SD	25.36	66.19	101.36
VL*	$\bar{x}$	127.778	162.556	151.714
	SD	37.76	66.57	79.29
RF	$\bar{x}$	130.111	152.11	157.571
	SD	23.94	41.74	46.79
QF	$\bar{x}$	127.926	159.667	157.000
	SD	29.02	58.16	75.80

*significantly different from isometric peak activity

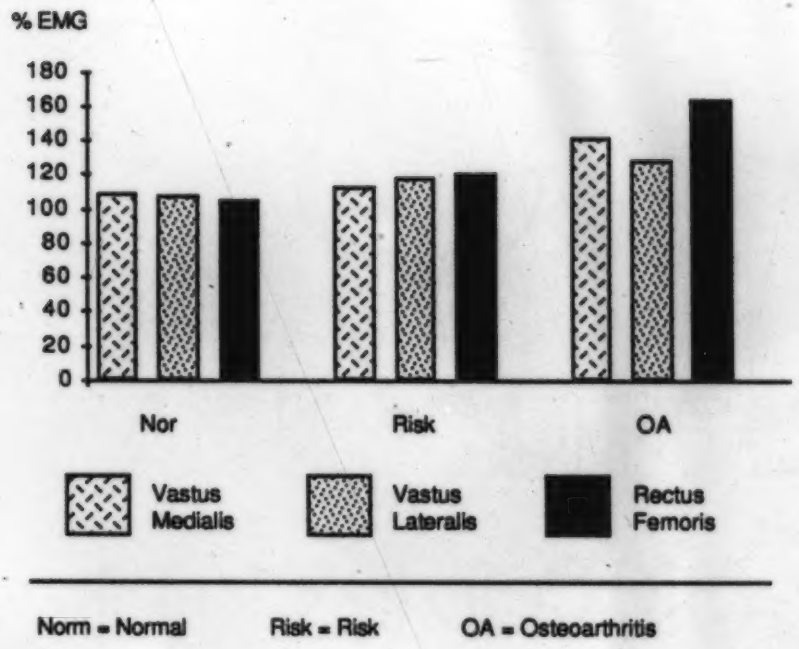

Figure 4.1 Comparison of normalised Isometric peak EMG

In terms of statistical significance, RF activity at 30 degrees knee flexion recorded by arthritic subjects, differed significantly from values recorded by the normal and risk groups ($p=.001$), as did total QF muscle activity ($p=.001$). Movement at fast speed had a significant effect over slower speeds of contraction with respect to VL muscle ($p=.03$) but was not significant with respect to VM and RF. In addition, the total RF activity occurring during slow and fast movements in arthritic subjects was significantly greater ($p=.0026$).

Figures 4.1 and 4.2 demonstrate mean and standard error values amongst the three study groups with respect to the quadriceps components evaluated during both isometric and isokinetic contractions. The trends exhibited by all groups under isometric and isokinetic conditions, previously described can be verified by observation of these figures.

In arthritic subjects, peak values recorded during isometric contraction (figure 4.1) show heightened activity of the VM and RF components, but a lower VL peak. This trend is also noted to occur under isokinetic conditions. The high VM values may reflect the heightened activity of this muscle as reported by Brucini (36), while the lower peak values recorded for VL activity support the view of Maquet (133) of between association of lateral thigh muscle weakness and varus malalignment. Additionally, Andrews and

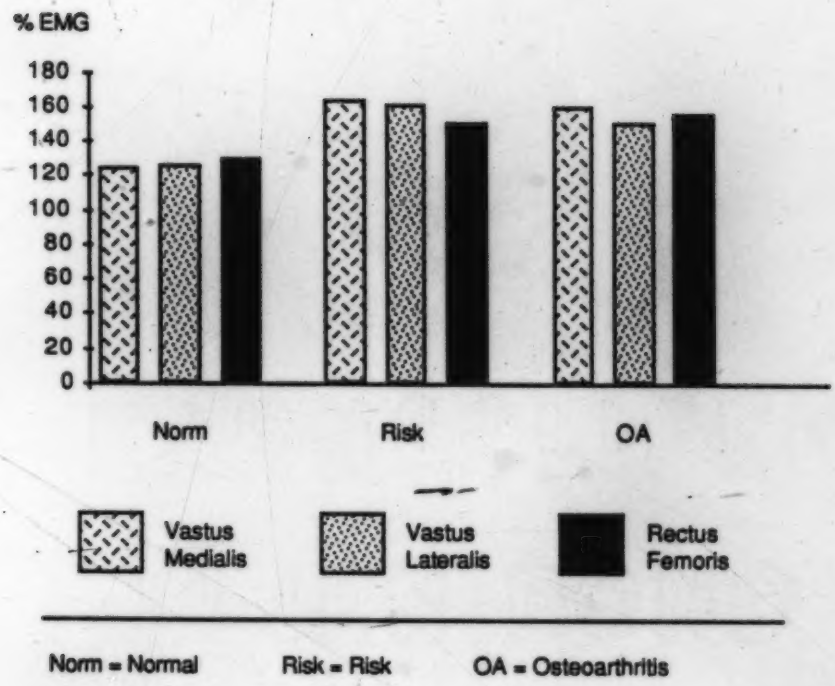

Figure 4.2. Comparison of normalised Isokinetic peak EMG

associates (251) noted higher VM and lower VL values in six females presenting with chondromalacia patellae tested under similar isokinetic conditions.

In this regard Richardson (255) studied the activity pattern of the knee joint in persons with patello-femoral syndrome (PFS) recorded during increasing speeds of ballistic activity. In accordance with findings in this study, Richardson (255) noted that while VM medialis activity in normals showed no marked increase with increasing speed of motion, VM increased its activity to a marked extent with increasing speed of movement in subjects with PFS. Richardson (255) attributed these observations to loss of tonic activity by the VM in subjects with PFS. Although stereotyped orderly recruitment appears to be most common (302), Harding (85) suggested that an arthritic joint produced an increased number of impulses or abnormal impulses, rendering the spinal centres abnormally excitable. Henneman and Olsen (174) described an increased firing pattern of motor units as a result of muscle disuse and Ianuzzo (256) found an increased firing pattern in relation to disuse at increasing speeds of movement. Cremer et al (302) suggest that some mechanism may be generally available to selectively activate motor units most adapted to the constraints imposed on the motor neuron pool by the movement pattern. In contrast to the findings of Richardson (255), results of this study indicate that the

VL muscle also displayed increased activity in risk and osteoarthritic subjects during fast movement. However, reported atrophy of slow twitch fibre loss in the VL occurring in OA (84) may alter the mechanism of recruitment occurring during sudden slow speed contractions (232), causing the movement to be initiated by a phasic unit, thus by-passing the slow-twitch motor unit mechanism.

If increased VM and VL activity in risk and arthritic subjects during fast contraction reflects some loss of tonic control during dynamic activity, problems of instability during gait might largely be accounted for on this basis.

While RF activity at higher speeds did not follow the expected trend of increased activity in this respect, RF activity in normal subjects showed increased activity during fast speed contractions, in accordance with Richardsons earlier findings (197). The discrepency of peak EMG activity recorded during slow and fast movements in the arthritic group may reflect fast-twitch fibre loss (261), although there is no documented evidence of fibre atrophy occurring within the RF muscle with respect to OA.

The significantly greater peak activity amongst all groups with regard to isokinetic exercise with the exception of RF, suggests that type of contraction is a major factor affecting the recruitment of motor units. Rosenzweig and Hinson (258) confirm the observation that

isokinetic contractions generally elicit significantly greater muscle action potentials than those evoked during isotonic or isometric contractions. Additionally, pain during isometric contraction, may yield higher normalised values of EMG activity in risk and OA groups during isokinetic contraction, since this movement may be the more comfortable. If subjects have some concomitant signs of patellofemoral dysfunction, as Kiss et al (252) suggest, this would compromise the ability of subjects to generate torque in a flexed position, since patellar ligament forces reach a maximum at 60 degrees (294).

The high peak values recorded at 90 degrees/sec in both risk and arthritic groups relative to normals, may also indicate that these subjects do not respond to velocity in the same manner as normal subjects (261). Changes in muscle fibre content in arthritics (83,84), producing altered patterns of recruitment and discharge frequency of motor units, may account for the high peak activity noted in these subjects as discussed above. The trend shown by risk subjects in this regard, may indicate the onset of similar changes in this group.

Additionally, to account for the relative high peak values recorded by arthritic subjects, it may be helpful to examine the effects of joint pathology on the ability of these persons to perform optimally during the normalising contraction. These observations may also shed light upon

the extent and nature of the muscle and joint dysfunction occurring in these subjects.

While Brownstein and associates (257) suggest that variation in the test range of motion may normally yield significantly different results particularly with respect to the VM and VL muscles, the muscle activity knee-angle dependence also reflects a relationship between the mechanical efficiency of the knee joint musculature and knee flexion (199). In general, flexion has the effect of increasing the quadriceps lever arm and shortening the hamstring lever arm. It is likely therefore that the inability to generate adequate EMG activity and torque levels at 60 degrees flexion reflects the presence of length changes in the quadriceps mechanism. Since EMG activity is length dependent (166), showing decreasing activity at longer muscle lengths (168), changes in the length or lever of the QF muscle group may result in muscular insufficiency (166). For example, a tight RF shortened as a consequence of pathology (23,258,295) may reduce peak normalising values of RF activity at 60 degrees knee flexion since the muscle may be relatively lengthened in this position. There also appears to be an optimal sarcomere length at which tension development is maximal (296). Above and below this length, tension is reduced (296), shortened muscle generating lowered torque values (259). Sahrmann (260) suggests a muscle will test weaker

if placed at a length in which the overlap of intracellular filaments are compromised. In contrast, at 30 degrees flexion, a shortened RF muscle might be in a more neutral or relatively shortened position. Eloranta and Komi (199) also report that in a sitting posture the RF generally reaches its peak activity towards the fully the extended position. Andriacchi and associates (199) found muscle response to be dependent on joint angle with highest muscle activity occurring when the knee musculature was shortened, with diminished activity when muscle was lengthened. Since higher values of EMG activity are evoked at shorter muscle lengths (168,183,199), and since RF would likely be less compromised at this angle a relatively high peak EMG value might be recorded with respect to the normalising value at 60 degrees flexion. Additionally to serve as a compensatory mechanism for decreased knee extension capability at 30 degrees flexion, the RF muscle may demonstrate heightened synergistic hip activity, recording proportionately high EMG values at this angle as result (24,261).

The muscle activity pattern in an unstable knee joint may also compromise ability to produce effective force production under some circumstances. For instance, Solomonow (262) noted a range of 37 to 46 degrees knee flexion produced a tendency to quadriceps torque failure in persons with a history of anterior cruciate ligament

deficiency. In fact, direct stress to the anterior cruciate ligament in this position of flexion, produced a moderate inhibitory effect on QF activity as recorded by surface EMG. Since the osteoarthritic knee joint is characterised by ligamentous instability (2,4), a similar response incurred during QF contraction at 60 degrees flexion in arthritic knees may produce lowered QF torque and EMG activity values in these subjects as a consequence. In contrast, Morrison (73) noted that varus and valgus laxity were reduced as the knee approached the extended position, possibly decreasing the need for hamstring co-activation. Therefore relatively high torque values might be recorded at 30 degrees knee flexion as a result. EMG values might also be high since the higher levels of EMG occur at shorter muscle lengths (168,183).

Haffajee and co-workers (166) noted the flexion angle for maximum torque to be about 50 degrees. This is of practical significance since weight-bearing activities in daily activities often occur in a semi-flexed position of the knee joint. The functional problems incurred by study subjects with respect to activities requiring the use of this angle, such as ascending or descending stairs, suggests the likelihood of relatively decreased torque capability at 60 degrees knee flexion under isometric conditions. Additionally, Grote (201) reported that the final centre of pressure was located more posteriorly in

osteoarthritic females than normals, suggesting that hamstring activation is heightened in an effort to prevent weight from falling anterior to the knee during activity. These findings indicate that strength performance of the QF at 60 degrees knee flexion may be relatively compromised in OA subjects as a result.

Additionally, Andriacchi et al (293) studying the effect of knee ligament dysfunction on functional ability, noted that sacrifice of the posterior cruciate ligament substantially reduced the quadriceps lever arm in the critical range of knee flexion required for stair climbing, (that is between 60 and 90 degrees). This observation is important since stair climbing is a functional activity which is considerably compromised in the arthritic subject and would explain a deficit in function at 60 degrees in subjects displaying similar functional problems.

b) Raw EMG activity patterns

Activity patterns for all groups under all contraction conditions are shown in plates 4.1 to 4.9. Chosen at random to depict activity patterns amongst the three groups, these graphs display decreasing amplitudes of raw EMG activity when risk and arthritic subjects are compared to the normal controls subjects. However in order to compare activity patterns of EMG across muscles and

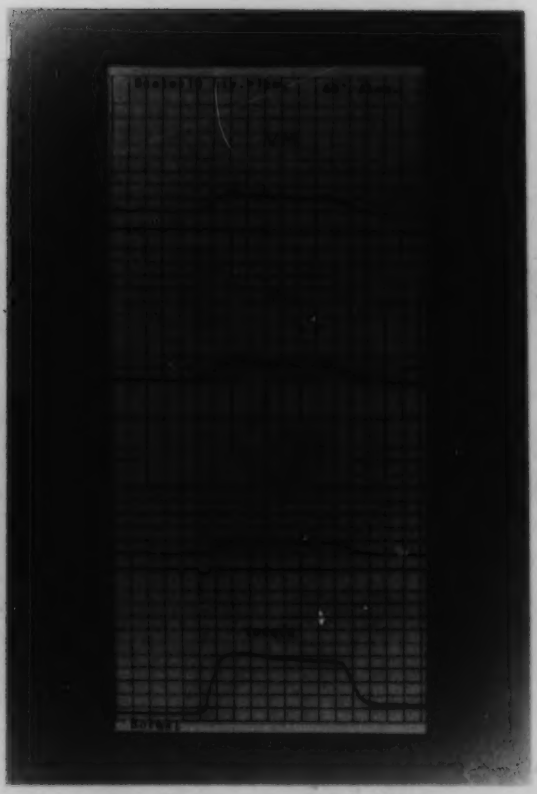

Plate 4.1. EMG and Torque of normal subject at 60 degrees knee flexion during isometric contraction

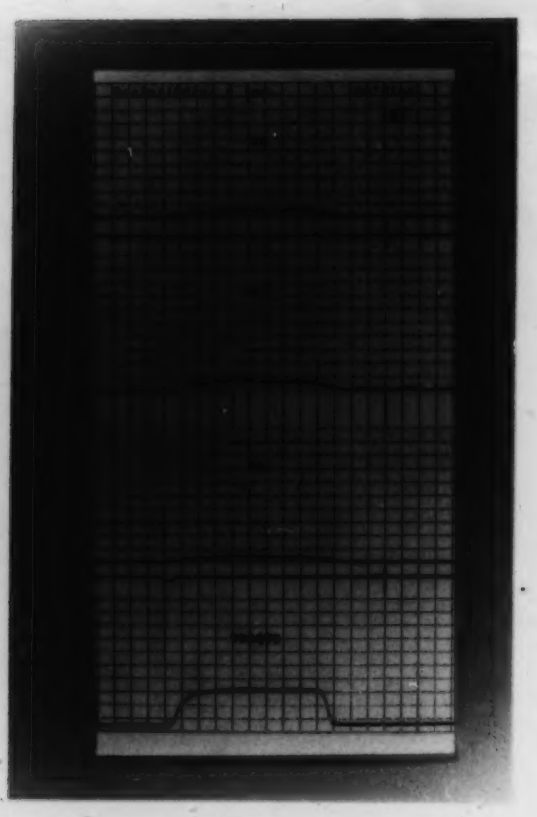

Plate 4.2. EMG and Torque record of risk subject at 60 degrees flexion during isometric contraction

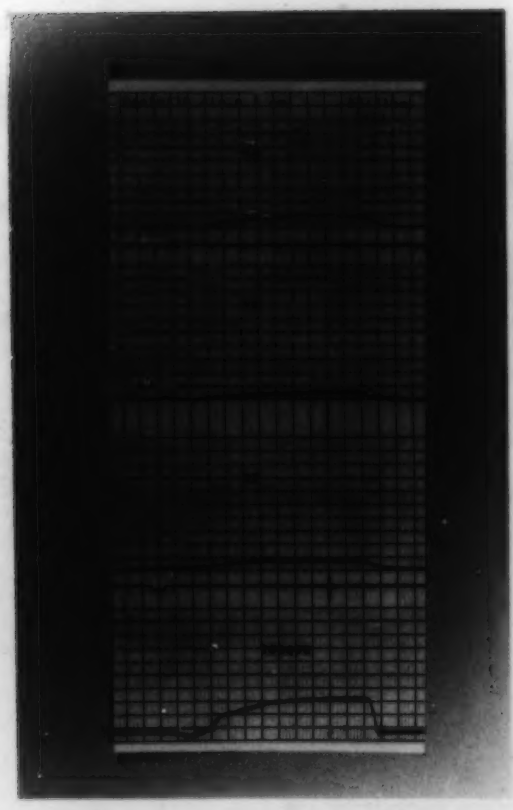

Plate 4.3. EMG and Torque record of OA subject during isometric contraction at 60 degrees flexion

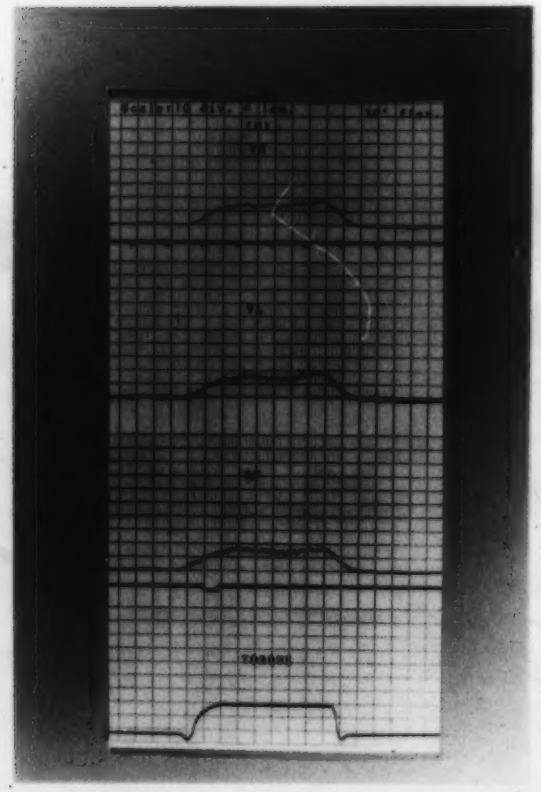

Plate 4.4. EMG and Torque of normal subject during isometric contraction at 30 degrees flexion

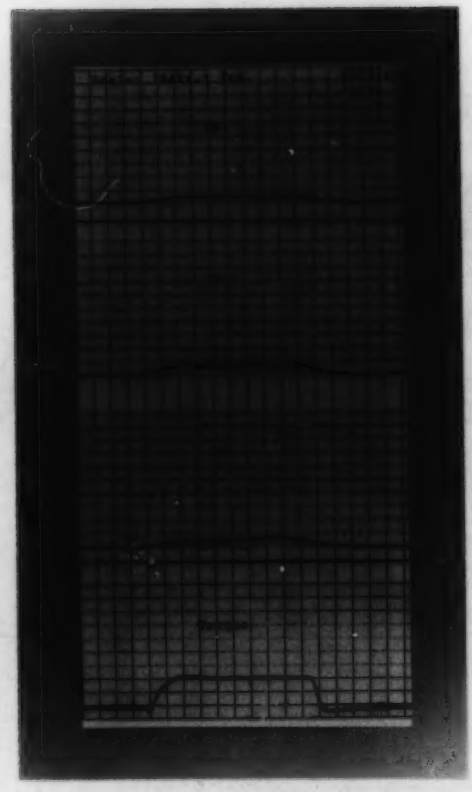

Plate 4.5.EMG and Torque of risk subject during isometric contraction at 30 degrees knee flexion

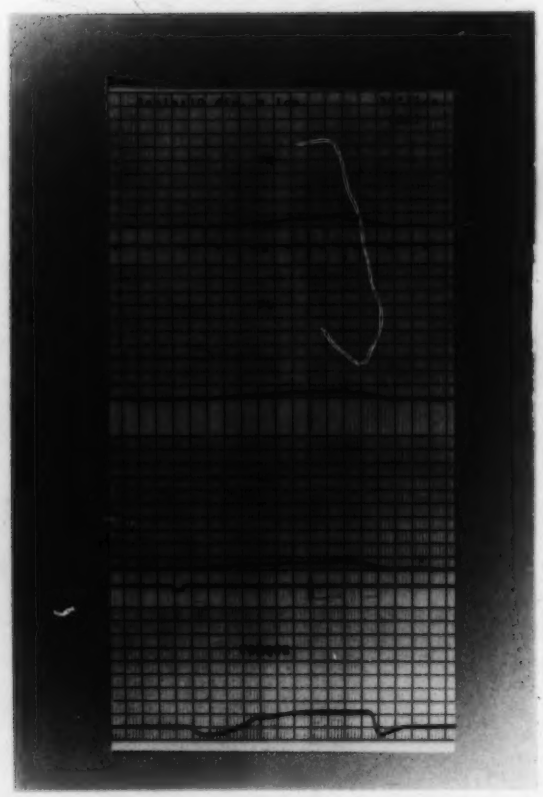

Plate 4.6. EMG and Torque record during isometric contraction at 30 degrees flexion

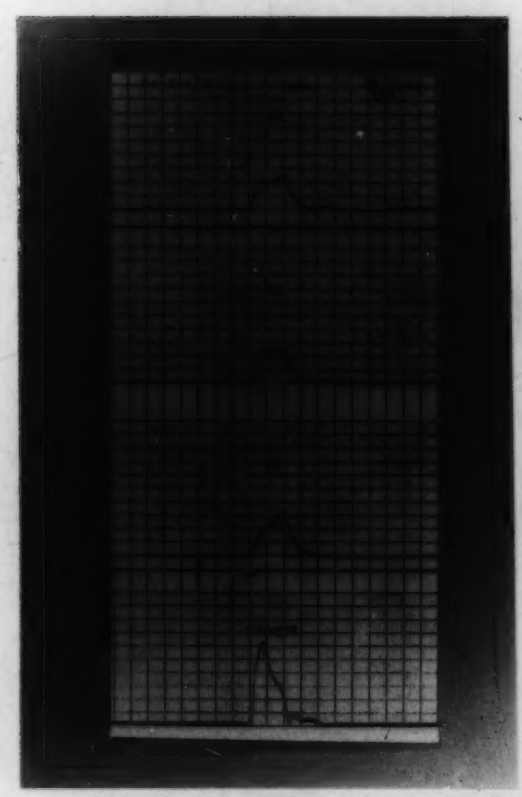

Plate 4.7. EMG and Torque record of normal subject during isokinetic contraction at 90 degrees/sec

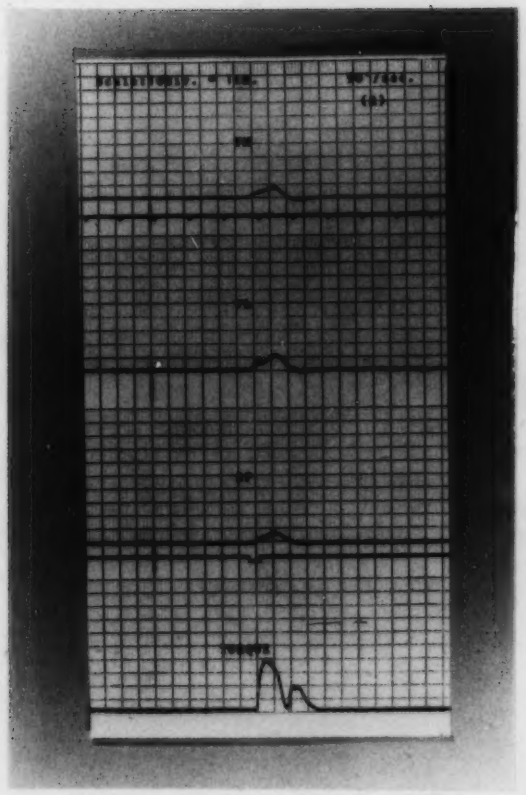

Plate 4.8. EMG and Torque record of risk subject during isokinetic contraction at 90 degrees/sec

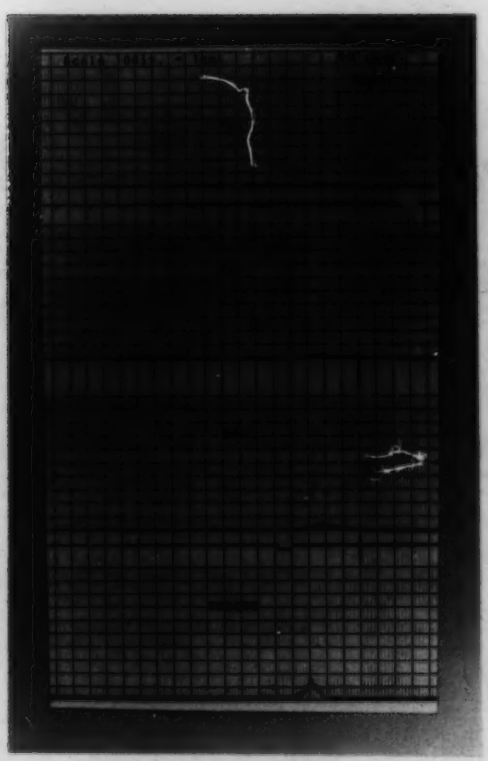

Plate 4.9. EMG and Torque record of OA subject during isokinetic contraction at 90 degrees/sec

groups data must normally be subjected to normalisation procedures if conclusions about EMG activity are to be derived.

c) IEMG

This value was defined as the area under the curve measured in the first second of the contraction period during both isometric and isokinetic conditions. This value was determined manually by counting the number of squares under the linear envelope in the first second of the contraction period. The IEMG was expressed in mm/sec and values were subject to the normalising procedure previously described. Intra-rater reliability made on manual IEMG calculations yielded a value r=.99. These values represent an indirect measure of the muscular activity produced during the first second of activity. Since the initial rate and amount of tension development are important considerations in defining the role of muscular change in OA, IEMG values calculated in the first second of contraction were used to provide insight in this regard, possibly providing more pertinent imformation than similar calculations measured over the entire contraction period. Santavirta (263) noted that due to the possibility of early fatigue in pathological conditions, the duration of contraction investigated should be relatively short.

Figures 4.3 and 4.4 provide graphic representation of

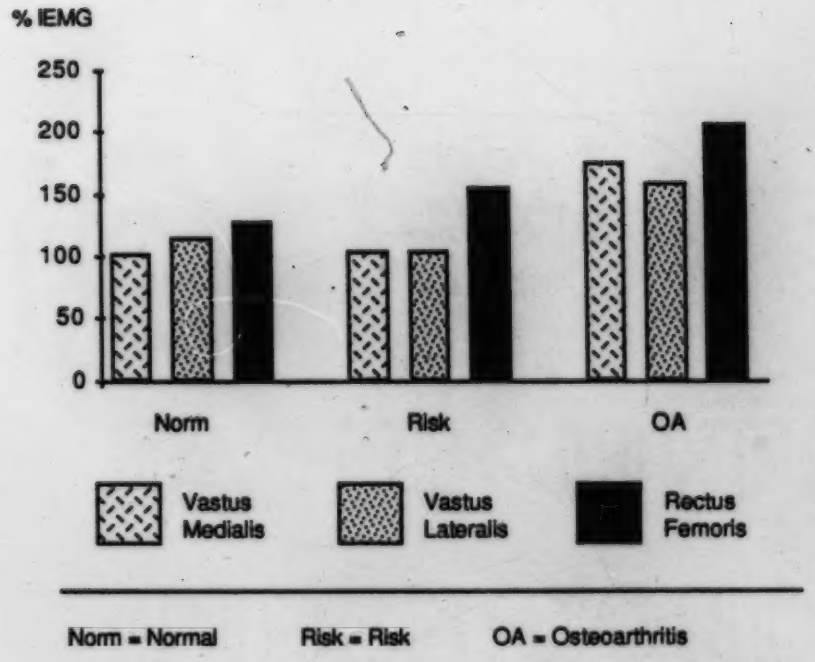

Figure 4.3. Comparison of normalised Isometric IEMG

mean differences found amongst study and muscle groups with respect to IEMG measures. Patterns produced during slow and fast movements are clearly different with regard to risk and osteoarthritic groups, but appear relatively consistent within the normal subjects. Here VM activity is lowest and RF activity highest irrespective of speed of contraction. Risk subjects also show a similar trend during isometric activity, but this pattern is altered slightly during isokinetic activity where subjects show high gains in VL activity.

TABLE 4.4: ISOMETRIC IEMG AT 30 DEGREES KNEE FLEXION

Mean ($\bar{x}$) and standard deviation (SD) of normalised values

		GROUP		
MUSCLE	STAT	NORMAL	RISK	OSTEOARTHRITIC
VM	$\bar{x}$	102.333	105.111	175.143
	SD	18.77	41.76	148.79
VL	$\bar{x}$	115.444	105.111	160.429
	SD	52.15	9.62	169.29
RF**	$\bar{x}$	129.000	155.333	207.571
	SD	42.42	122.25	169.29
QF	$\bar{x}$	115.592	125.481	181.048*
	SD	37.78	71.21	162.45

*significantly different from risk and normal

**significantly different from VL AND RF

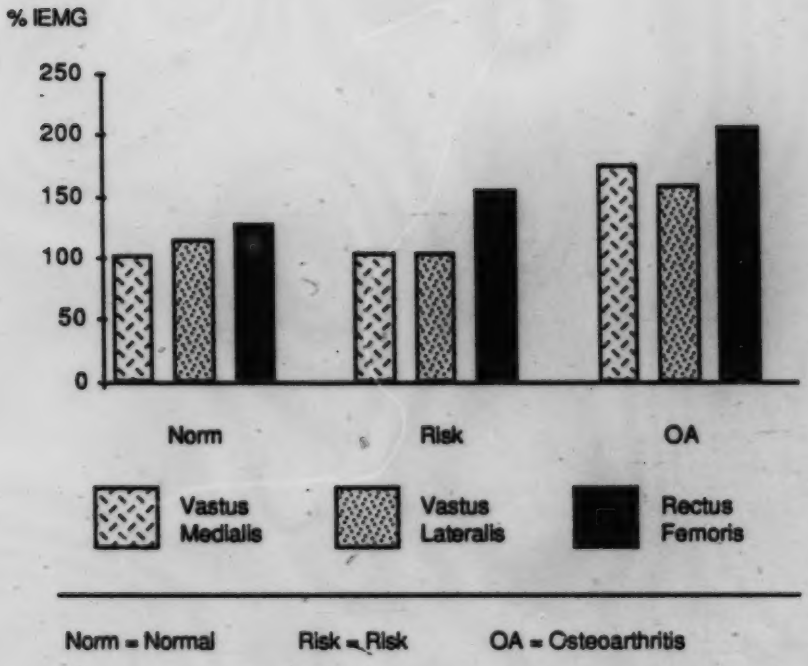

Figure 4.4. Comparison of normalised Isokinetic IEMG

TABLE 4.5: ISOKINETIC IEMG for 90 degrees/sec

Mean values ($\bar{x}$) and standard deviations (SD) of normalised values

		GROUP		
MUSCLES	STAT	NORMAL	RISK	OSTEOARTHRITIC
VM*	$\bar{x}$	168.330	212.778	211.000
	SD	54.71	97.92	190.55
VL*	$\bar{x}$	184.000	233.778	220.00
	SD	55.94	118.06	183.52
RF*	$\bar{x}$	216.444	237.222	294.00
	SD	97.15	150.33	196.38
QF*	$\bar{x}$	189.591	227.926	241.667
	SD	39.80	122.100	191.81

*significantly different from isometric values

With reference to figure 4.3 and table 4.4., IEMG activity of VM and RF recorded during isometric contraction is notably higher in arthritic subjects, although this difference is not statistically significant. During isokinetic contraction, increased VL activity is also noted in these subjects. However, these values are not as high as those exhibited by the risk subjects at fast speed. Analysis of individual patterns of muscle activity amongst the three groups show a distinct trend by risk subjects to

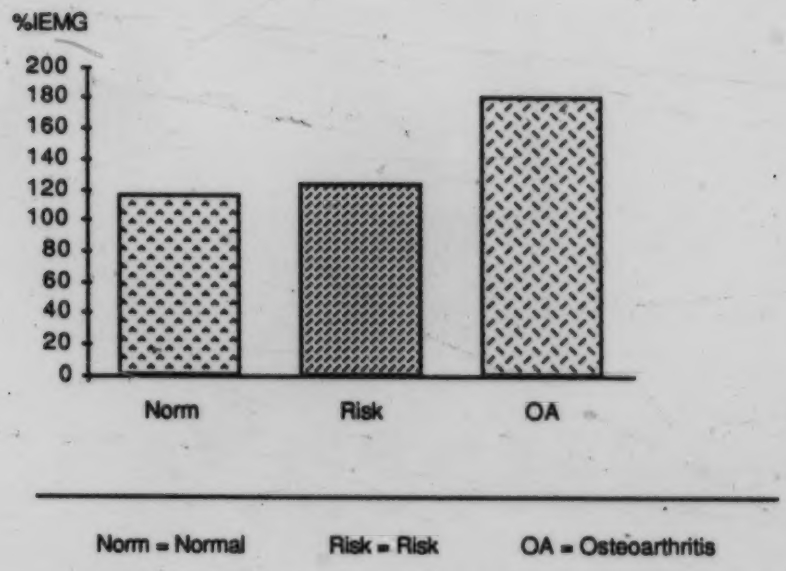

Figure 4.5. Comparison of normalised Isometric IEMG (QF)

increased RF activity at 30 degrees flexion and activity is increased in all muscle groups during isokinetic contraction. Increasing IEMG values, significant at the p =.02 level (VM and RF) and p=.0069 (VL) were noted at fast speeds, emphasising speed of movement as a major influence on motor recruitment. Eloranta and Komi (198) also found the IEMG values of VL and VM tended to increase with increasing speeds of movement. Additionally, RF muscle activity was significantly higher (p=.0273) in terms of IEMG activity compared amongst the three muscle groups. Eloranta and Komi (198) noted no marked differences in RF activity to occur at fast and slow speeds although RF activity was increased in all three groups at fast speeds in accordance with Richardson (197). Arthritic subjects also demonstrated significantly higher values of total QF IEMG activity (p=.0052) during isometric contraction at 30 degrees flexion (figure 4.5).

While the electrical activity generated by a muscle may be influenced by a number of factors, the IEMG has been found to provide a relatively efficient tool in assessing the contribution of a specific muscle or group of muscles in a given experiment (169). In this study, IEMG activity appeared to adequately differentiate activity levels between muscles and amongst subjects. The high IEMG values noted overall by arthritic subjects with regard to activity generated at 30 degrees knee flexion, may substantiate the

claim by Grote (201) that osteoarthritic muscle is required to generate substantial activity in an effort to stabilise the affected joint and prevent the knee from collapsing. In fact, Santavirta (263) found a relatively high IEMG activity was generated by the VM muscle in men during the post menisectomy period, since the VM muscle was required to generate a significantly higher activity to produce the same force as the control leg. Denham et al (264) note that the force transmitted in one compartment of the knee joint is the sum of the body weight, the combined flexor and extensor muscle action and the excessive tension that the ligaments undergo in maintaining the articulation. Additionally, greater tension must often be generated in the presence of tibial malalignment if subluxation is to be prevented. McLeod (cited in 263) suggested that evidence of high VM IEMG activity was indicative of the need to stabilise the patella. Compared to normal subjects, Smidt et al (265) found subjects with degenerative knee arthritis spent an excessive amount of time in the stance phase of the involved side and preferred to maximise the time for the foot flat portion of stance. This provides a measure of stability during gait and may require increased EMG to be generated by the QF muscle as a result. Andriacchi et al (293) noted substantially higher loads can be generated at the knee joint due to increased quadriceps force when the mechanical efficiency of the quadriceps mechanism is

compromised. Peat et al (150) reported that continuous quadriceps activity throughout the stance phase of gait was required to stabilise the flexed knee in arthritic subjects, indicating the additional demand placed on the quadriceps musculature in these individuals.

In normal individuals the rotary force at the knee joint is lessened as the subject extends to the neutral position. Thus low levels of VM and VL activity are generally found during the last thirty degrees of distal segment stabilised extension (266). Low levels of activity displayed by normal and risk subjects confirm this observation. However, osteoarthritic subjects displayed considerably increased IEMG activity at this angle emphasising the increased need to stabilise the knee in this position. This finding may be of consequence, since patella subluxation commonly occurs at this angle (266). This would support McLeod (cited in 263) as stated above. Others note that increases in joint stability may be attained by increasing the activity and number of active muscles resulting in increases of over 400% by activation of the appropiate musculature (267). Goldfuss (132) reported that knee abduction and adduction were limited significantly when the knee musculature was pretensed. Activation of this mechanism would likely limit the extent of instability in subjects with gonarthrosis since when quadriceps activity is high, a large compressive force is

produced across the joint, effectively stabilising against varus and valgus stresses (199).

The implications of high VM and VL activity occurring in both risk and osteoarthritic groups during fast movements have been discussed with reference to the work of Richardson (197) in the previous section. Differences in EMG activation in arthritic sujects may also reflect the observation by Collopy et al (268), who found osteoarthritic patients differed from normal in that their knees were in too much flexion during stance and too little flexion during the swing phase of gait. The extreme variability noted with respect to the risk and arthritic subjects in the measurement of these data, precludes definitive conclusions. However, the trends exibited by the risk group in direction of the arthritic subjects are striking.

c) IEMG/torque data

In this analysis, the IEMG values were those calculated during the first second of the contraction phase under both isometric and isokinetic conditions. Torque values noted during the first second of torque generation were calculated by measuring the area under the torque curve registered over this time period. Normalized values were used in the analysis and the relationship between the IEMG and torque was calculated by dividing the IEMG values

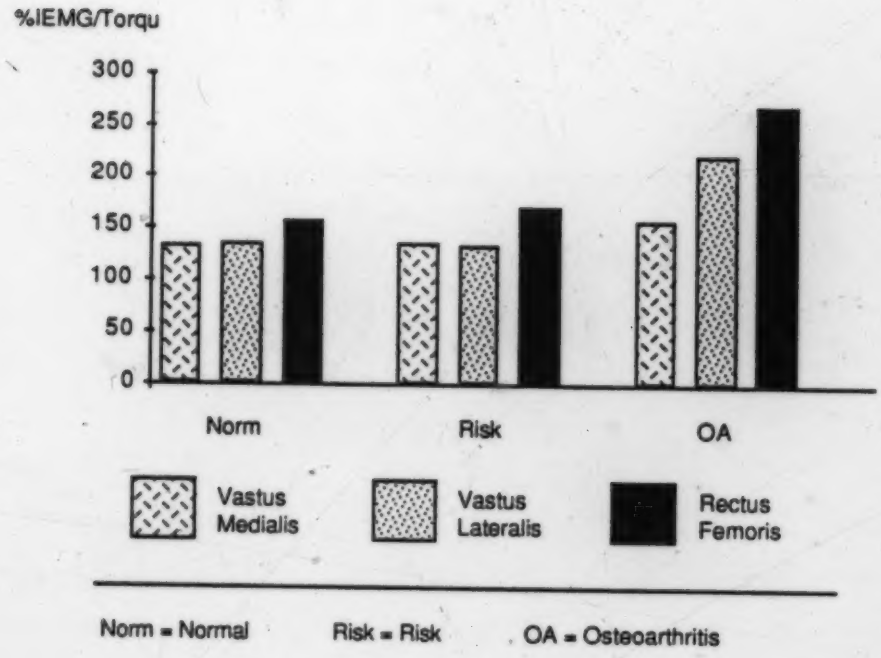

Figure 4.6. Comparison of normalised Isometric IEMG/torque

by the respective force measures. The IEMG/torque ratio has been described as a measure of muscular efficiency by De Vries (cited in 292). In this study the ratio between the IEMG and the torque derived during their respective first seconds of activation, provided a means of evaluating the relative level of motor capability amongst three differing groups, although EMD time was not accounted for.

Graphic representation of IEMG/torque values is displayed in figures 4.6 and 4.7.

TABLE 4.6: ISOMETRIC IEMG/TORQUE ratio-30 DEGREES FLEXION

Means ($\bar{x}$) and standard deviation (SD) of normalising value

		GROUPS		
MUSCLE	STAT	NORMAL	RISK	OSTEOARTHRITIC
VM	$\bar{x}$	135.55	137.44	159.33
	SD	68.16	37.63	146.33
VL	$\bar{x}$	137.44	135.33	221.00
	SD	69.38	53.65	166.75
RF	$\bar{x}$	159.33	171.88	269.85
	SD	75.89	95.26	170.75
QF	$\bar{x}$	144.11	148.48	236.38*
	SD	71.14	72.18	161.27

*significantly different from normal and risk

These data show the following. Firstly, at 30 degrees knee flexion, higher IEMG/torque ratios are recorded for arthritic subjects, with RF displaying considerably higher mean values than VM and VL. In general, however, with the exception of higher mean values recorded for individual muscle activity for the arthritic group, all three groups show the same pattern of response in terms of overall QF muscle activity. That is, there is equivalance amongst VM and VL with respect to IEMG/torque ratios recorded in all three groups and in addition, these ratios are generally lower than comparitive recordings of the RF muscle.

TABLE 4.7: ISOKINETIC IEMG/TORQUE ratio at 90 DEGREES/S

Means ($\bar{x}$) and standard deviation (SD) of normalising value

		GROUPS		
MUSCLE	STAT	NORMAL	RISK	OSTEOARTHRITIC
VM	$\bar{x}$	141.11	238.55	338.5
	SD	61.46	191.61	491.83
VL	$\bar{x}$	163.00	214.66	215.33
	SD	93.37	86.75	336.72
RF	$\bar{x}$	180.44	237.77	263.83
	SD	98.13	103.84	226.44
QF	$\bar{x}$	161.51	230.33	272.55
	SD	84.32	100.07	239.42

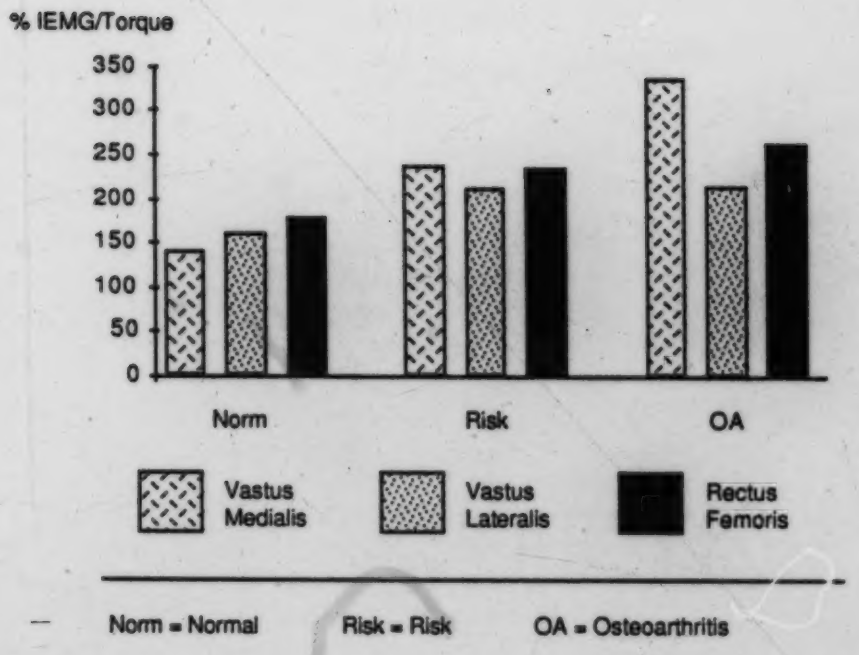

Figure 4.7. Comparison of normalised Isokinetic IEMG/torque

Differences between arthritic and normal and risk groups were noted with respect to QF IEMG/torque ratios during isometric contraction. These values were significant at p=.0035 level. No significant differences were noted amongst muscles in this regard, although distinctly higher values were recorded for the RF muscle as indicated in Table 4.6. The values for muscle effect across all three groups (159.680 (VM); 160.080 (VL) and 194.800 (RF) indicate relatively consistent measures of IEMG/torque ratios occurring between VM and VL during the initial stages of contraction in all three groups. This finding is of interest since the literature indicates the importance of VM/VL imbalance as a factor in knee joint dysfunction (291). Eloranta and Komi (199) studying postural effects on the function of the quadriceps muscle under concentric contractions, found that the vasti muscles, functioning only around the knee joint, demonstrated a plateau type activity in the slowest contraction speed in a seated position. The low IEMG/torque ratios recorded by both normal and risk groups in this study implies that a highly efficient force is produced during the initial stages of an isometric muscle contraction under normal circumstances. Low IEMG/torque ratios are also associated with increased power production (199). This is important since the QF muscle is responsible for providing an important stabilising function across the knee joint, especially

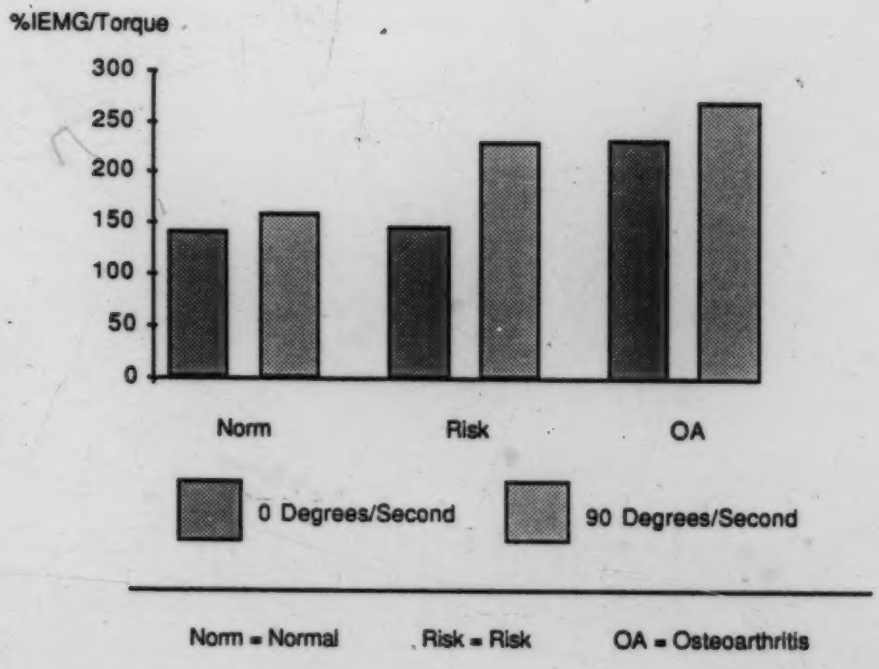

Figure 4.8.Comparison of IEMG/torque Isometric and Isokinetic

during terminal extension (199). The higher IEMG/torque values recorded by osteoarthritic subjects may indicate a less effective attempt to stabilize the joint under similar conditions.

During fast concentric contractions, the IEMG/torque ratios were greater, particularly with respect to risk and osteoarthritic subjects (figure 4.8). No significant difference was noted amongst muscles or groups in this regard. Viitasalo (271) attributed the effect of speed on IEMG/torque ratios to differences in the motor recruitment pattern and suggested that differences between IEMG/torque ratios reflect the decreased amount of force in spite of high neural output. This in turn is explained by the reduction of effective time for single actinmyosin cross-bridges to produce force. However at the relatively low speed of 90 degrees/sec this effect should be reduced. In fact observation of the speed effect on IEMG/torque ratios in normal subjects show very small differences between values recorded during slow and fast speed movements. Mean IEMG/torque values recorded at 90 degrees/sec show risk and osteoarthritic subjects produced higher mean values than the normal group with respect to recordings of all three muscles. Furthermore VM and RF ratios in the osteoarthritic group are notably higher than those produced by the risk group and VM displays higher values than RF. VL ratios in both groups however are almost of equivalent value. These

findings indicate that with the exception of individuals in the normal group, subjects in the risk and arthritic groups display decreasingly effective muscular activity during isokinetic contraction, with VM in the arthritic group producing the least effective contraction. As stated previously, a high IEMG/torque value is likely indicative of two factors, firstly increased recruitment and secondly force reduction, although the latter was not evident in this study. During dynamic contraction Richardson (255) showed the heightened phasic muscle activity of VM in persons with PFS. Similarly in this study, increases in VM activation during dynamic movement appear to follow this trend producing relatively high IEMG values as a result.

D) TIME FACTORS

a) Time Rate of Tension Development-Isometric

The time rate of tension development (TRTD) required to attain 50 % of maximum torque capability at both 30 and 60 degrees flexion was calculated by measuring the height in mm. attained at 50% of peak torque divided by the time elapsed to reach this height. The point at which the torque curve began its incline from the baseline was defined as zero seconds. The TRTD was therefore the rate of force produced at 50% peak isometric contraction per unit time.

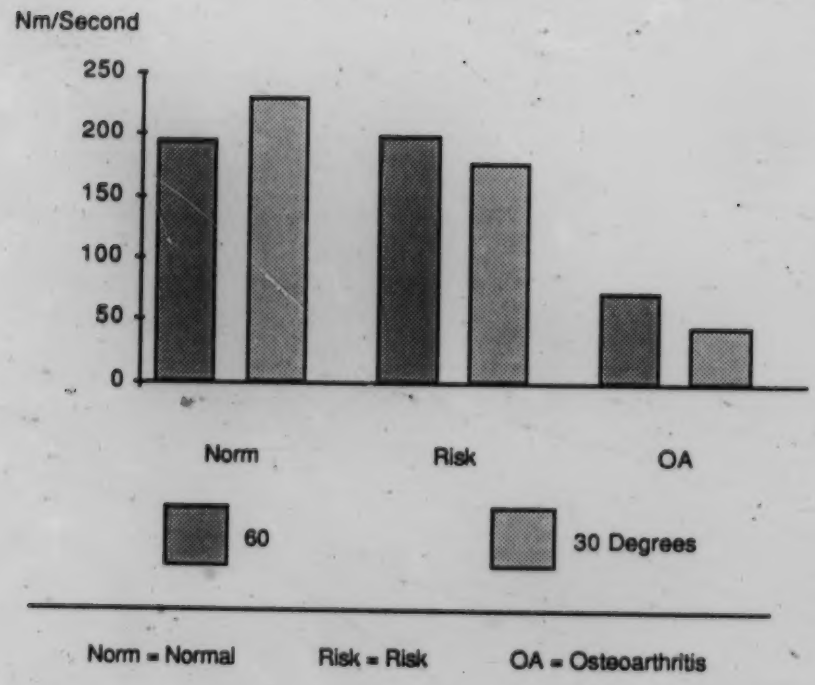

Figure 4.9. Comparison of Isometric TRTD at 50% torque max.

The data are expressed in Nm/sec.

Results of a two-way ANOVA used in the analysis are presented in table 4.8. Graphic representation of these data is shown in figure 4.9.

TABLE 4.8: ISOMETRIC TIME RATE OF TENSION DEVELOPMENT (TRTD) at 50% torque max

Means($\bar{x}$) and standard deviation (SD) values in Nm/sec

	STAT	NORMAL	RISK	OSTEOARTHRITIC
60 degrees	$\bar{x}$	196.98	200.85	74.14*
	SD	83.22	110.85	73.60
30 degrees	$\bar{x}$	230.64	178.48	46.71*
	SD	84.52	90.48	46.71

*significantly different from normal and risk

Observation of this data, shows the considerable reduction in rate of tension development in the arthritic group, both at 60 as well as 30 degrees flexion. This decrease was significant at the p=.001 level between groups as measured by a two-way ANOVA and at p=.009 and P=.0003 levels with respect to angulation at 60 and 30 degrees respectively as determined by one way ANOVA. At 60 degrees flexion, both normal and risk groups displayed similar levels of tension development, while the arthritic group

showed considerably lower values. At 30 degrees, the rate of tension development declined in risk and arthritic groups, but was increased to some degree in normals.

These results tend to confirm the importance of timing of muscle activity in maintaining the integrity of the joint as suggested by Radin (15,16). In particular, without appropiate rate of tension development, joint destruction would likely be assured.

During normal gait, the extensor muscles must be activated rapidly to prepare the limb for weight acceptance and tranfer. Failure to prepare for weight bearing may have deleterious consequences as described by Melvill Jones et al (19).

The relatively slow rate of tension development in arthritic subjects may reflect problems in motor recruitment occurring secondarily to changes in muscle fibre composition. Thorstensson (219) noted a high rate of tension development to correlate with a high percentage of fast-twitch fibres in the vastus lateralis muscle, indicating that the TRTD development may be influenced by the proportion of fast twitch fibres in a given muscle (286). Reported fast-twitch fibre loss occurring in gonarthrosis (83,84) could alter the rate of tension development in the knee extensors in accordance with the findings of this study. Bozec and Maton (272) also note that motor unit firing rate is related to the contractile

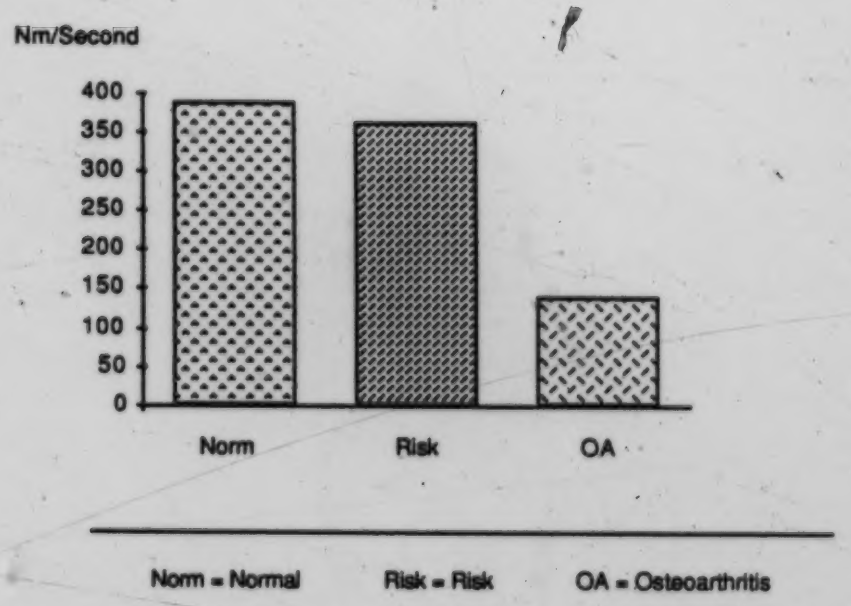

Figure 4.10. Comparison of TRTD during Isokinetic contraction

characteristics of the muscle and suggest muscles may be recruited according to the ratio of their type I and type II fibres. If both type I and type II fibre loss occur in gonarthrosis, as suggested by Glasberg and associates (84), it is likely that rate of tension development will be altered to a considerable extent as a consequence. Decreasing rates of tension development noted in both risk and arthritic subjects at 30 degrees of flexion may reflect the extent of prevailing joint instability, since the vasti must be capable of fixing the knee in extension, particularly during quick movement, if optimal approximation of the articulated bones is to occur (194).

b) Time Rate of Tension Development-Isokinetic

The time rate of tension development for isokinetic contractions was calculated by dividing the peak torque values, measured in mm. by the time period in tenths of seconds required to reach these values. The conversion factor 1mm= 3.88 Nm. was used in the analysis, and results are reported in Nm/sec. A summary of a one way ANOVA procedure used to analyse these data are presented in table 4.9. and figure 4.10.

Observation of the tabulated and graphic data presented in table 4.9 and figure 4.10 indicate the extent of the difference between the rate of tension development

in the normal, risk and arthritic groups. Here the arthritic group displayed significantly low rates of tension development (p=.000). Factors affecting rate of tension development have been discussed in the previous section. These include problems of motor recruitment in particular (272). It is also likely, that a deficit in tension development during isokinetic contractions, provides a better reflection of the magnitude of fast fibre loss occurring in these subjects, since these fibres will be recruited with increased velocity of motion. The low rate of tension generated by arthritic subjects at speeds equivelant to that employed during gait, are likely reflective of the reduced rate of knee motion in ambulation in conjunction with a decreased gait velocity reported in the literature (29,25).

TABLE 4.9: TIME RATE OF TENSION DEVELOPMENT in ISOKINETIC CONTRACTION

Means (x) and standard deviation (SD) values in Nm/sec

STAT	NORMAL	RISK	OSTEOARTHRITIC
$\bar{x}$	338.84	363.40	141.11*
SD	59.13	94.32	70.26

*significantly different from normal and risk

c) Rate of Initial Rise of Torque Curve-Isometric

The tangential slope of the torque curve was calculated by dividing the height in mm. measured at the confluence of the initial gradient and the torque curve, by the time required to reach this value. Measures were recorded during isometric contraction, at both 60 and 30 degrees flexion. These data were converted to Nm. and summarised in Table 4.10 and shown graphically in figure 4.11.

Findings are as follows. Firstly, there was a significant difference ($p=.0019$) between arthritic, normal and risk groups with respect to the initial rise of the torque slope. This difference was also significant for angle effect ($p=.005$) at 30 degrees flexion.

In addition, calculations of this value at both 60 and 30 degrees flexion, indicated a relative decline of the risk group compared to normal subjects. This finding may be indicative of an early change in the recruitment pattern as well as decreasing numbers of type II muscle fibres in risk subjects. In addition to factors previously discussed, the inhibitory influence of hamstrings on the ability of the quadriceps muscle to generate tension at slow speed and increasingly so at fast speed should not be discounted as a factor in influencing the timing of knee extensor activity in arthritic subjects and to a lesser extent in risk subjects. Reduced torque curves produced by subjects

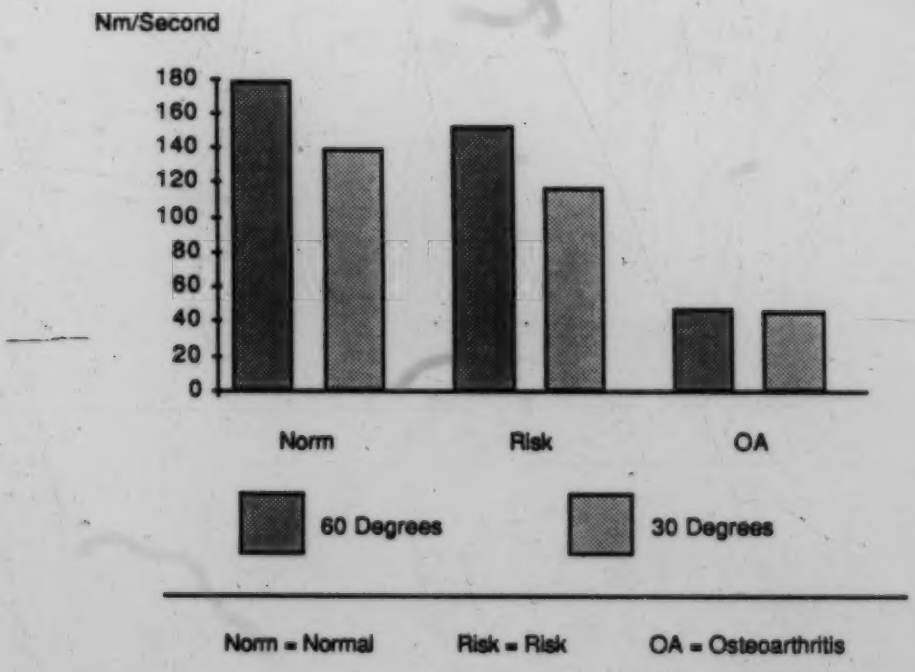

Figure 4.11 Comparison of Isometric TRTD

in both risk and arthritic groups at 30 degrees flexion, suggest the possible influence of hamstring activity on the ability of the quadriceps muscle to develop tension. Watkins et al (205) suggest that subjects sitting with their knees extended, also place the hamstring muscles in the lengthened position. Further elongation during rapid extension may activate the flexor stretch reflex, interfering with limb extension. Watkins et al (205) supported this idea by noting that electrical activity in the flexor muscles is increased by passive stretching in the sitting position.

TABLE 4.10:INITIAL RISE OF TORQUE CURVE-ISOMETRIC

Means ($\bar{x}$) and standard deviation (SD) values in Nm/sec

	STAT	NORMAL	RISK	OSTEOARTHRITIC
60 degrees	$\bar{x}$	179.76	153.88	48.26*
	SD	157.06	78.45	52.92
30 degrees	$\bar{x}$	140.10	117.68	47.84*
	SD	64.52	56.06	84.31

*significantly different from normal and risk

d) Electromechanical Delay (EMD)-Isokinetic

EMD was measured as the time in msecs. between the onset of EMG activity and the initiation of torque output. Activity was defined at that point above the baseline at which EMG and torque readings displayed deflection on the pen tracings. This value was calculated for all muscles in the three groups as time delays were variable amongst the QF components in all three groups. The results of a two way ANOVA are presented in table 4.11 and figure 4.12.

TABLE 4.11: ISOKINETIC EMD at 90 degrees per sec

Means ($\bar{x}$) and standard deviation (SD) values (msecs)

MUSCLE	STAT	NORMAL	RISK	OSTEOARTHRITIC
VM	x	164.4	121.0	182.0
	SD	119.3	111.5	129.6
VL	$\bar{x}$	154.4	127.0	182.0
	SD	128.8	109.7	129.6
RF	$\bar{x}$	51.1	74.4	182.0
	SD	40.7	163.1	126.9

Observation of these data show an overall increase in EMD in the arthritic subjects with respect to all muscle groups. In the normal and risk groups, EMD values are highest for VM and VL and lower for RF. Risk subjects

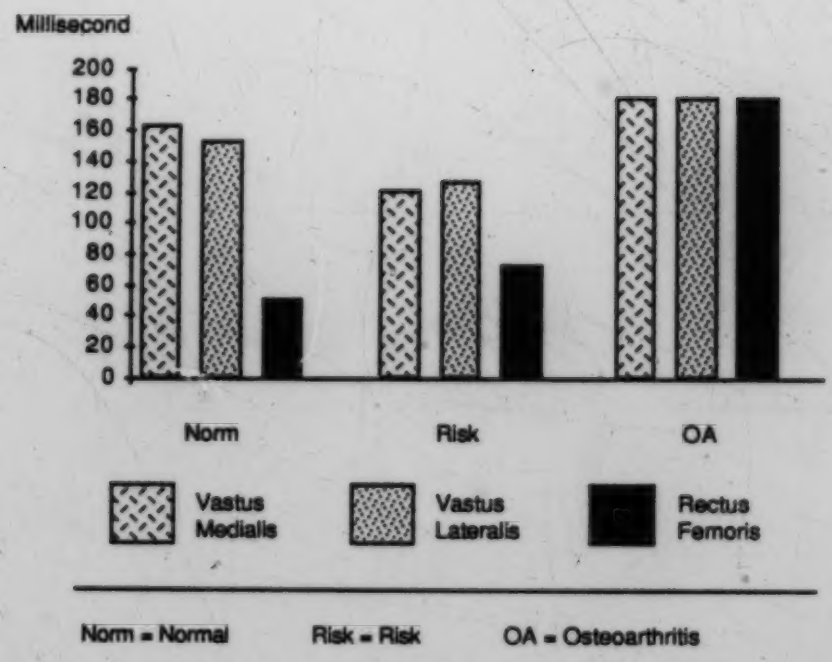

Figure 4.12. Comparison of Isokinetic EMD time

showed reduced values of EMD with respect to VM and VL compared to the normal group, indicating a faster response time for these muscles in this group. Findings were not significant amongst groups or muscles, although RF activity in the arthritic group showed a considerably higher mean value than the normal and risk groups ($p=.11$). In general the delay for all groups was higher than that reported in the literature (186,187). This may reflect methodologies employed both in the experimental protocol and in the data collection. The higher EMD values recorded by arthritic subjects may reflect altered motor unit composition and recruitment patterns and are possibly indicative of type II fibre loss (187). Although EMD values in risk subjects were lower than those recorded for normal subjects with respect to VM and VL, there was a longer delay noted for the RF muscle in the risk group . This finding may be indicate an early change in RF muscle reactivity in this group.

The increased EMD values in the arthritic group may emphasise the extent of timing deficit during gait reported by Smidt et al (265), who found the timing of foot placement to be asymmetrical in pre-operative assessment of subjects with degenerative arthritis of the knee joint.

e) Time to Peak (TTP) EMG - Isokinetic

Time to reach peak EMG during isokinetic movement was calculated in tenths of seconds between the onset of EMG

deflection from the baseline and the time period at which point peak EMG activity occurred. These data were subjected to a two way analysis of variance and the results are displayed in table 4.12 and figure 4.13. TTP EMG measures were recorded with the intent of establishing the total time variation amongst the three groups in attaining peak EMG activity. TTP measures showed the RF muscle to have a faster rise time to peak activity amongst all groups. Mean values did not however reach significant levels, ($p=.10$).

The extremely short period of time required for RF peak activation in arthritic subjects, may indicate the extent of adaptation in this muscle, since it is the shortest muscle of a synergistic group which is more readily recruited in a given movement pattern (24).

TABLE 4.12: Isokinetic time to peak (TTP) EMG activity

Means ($\bar{x}$) and standard deviation (SD) values in secs.

MUSCLE	STAT	NORMAL	RISK	OSTEOARTHRITIC
VM	$\bar{x}$	.86	.75	.86
	SD	.21	.15	.41
VL	$\bar{x}$	.75	.75	.66
	SD	.16	.14	.35
RF	$\bar{x}$	.71	.73	.53
	SD	.25	.13	.29

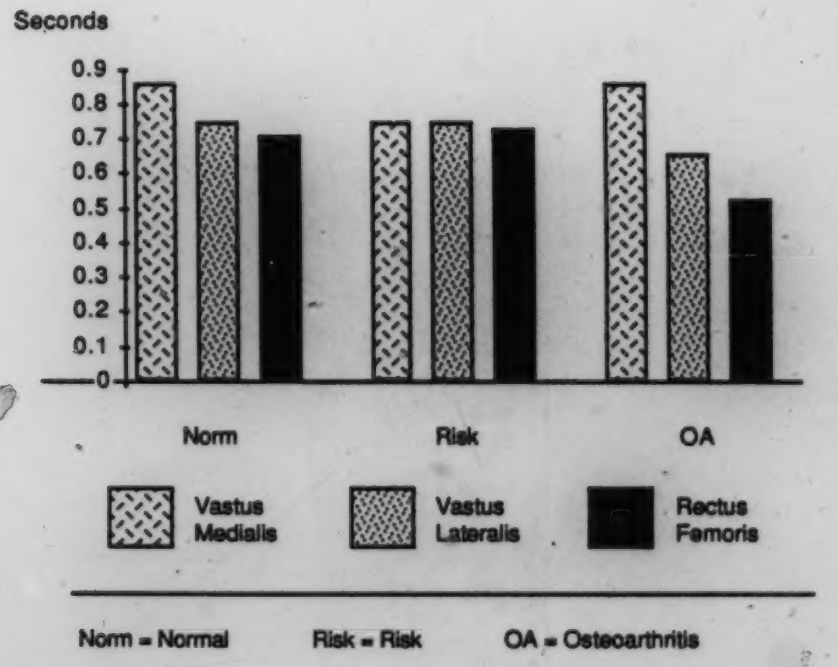

Figure 4.13. Comparison of Isokinetic TTP EMG

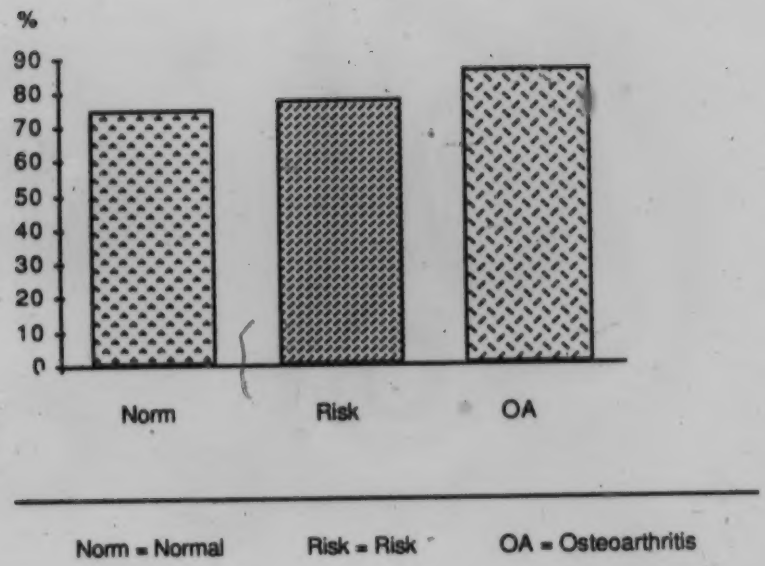

Figure 4.14. Comparison of normalised Isometric torque

In addition, although the timing of the VM and VL muscles are found to be relatively similar during gait under normal circumstances (196), there is a considerable deficit in the TTP for the VL muscle in the arthritic group in this study. This may signify a deficit in the reactivity of this muscle which has been implicated in the development of the varus knee (133). In this regard Murray et al (273) found functional deficit, alterations in stance time and marked extensor weakness after excision of the VL muscle, indicating that VL dysfunction may considerably alter the loading pattern of the knee joint. Since VL is the largest of the quadriceps heads, this effect may be considerable.

5) Torque Measures

a) Normalised Torque Values

Peak torque values, recorded during isometric contraction at 30 degrees flexion and isokinetic movement at 90 degrees/sec were normalised against the same values recorded at 60 degrees knee flexion. These values represented the highest recorded amplitude of torque activity above the baseline and were recorded in Nm. These data were analysed separately, using a one way ANOVA in each instance. These results are presented separately in tables 4.12 and 4.13 and graphically in figures 4.14 and 4.15.

While normal and risk groups displayed a 25% decrease in torque values recorded at 30 degrees knee flexion relative to 60 degrees knee flexion, arthritic subjects performed more ably at this angle with a percentage decline relative to 60 degrees knee flexion of 14%. The possible reasons for this discrepency have been discussed previously. These include factors such as the need to provide increased stability to the knee joint often rendered unstable by the disease process (201,293). In addition, the habitual use of the knee joint in the flexed position noted by several authors (30,150,265) may provide a training effect of the quadriceps muscle at this angle, resulting in relatively efficient contraction at 30 degrees (261). In general, however these findings are supported by the work of others (166,216,219), although the literature also supports the variability of flexion angle associated with optimal torque generation (215,222).

The higher percentage values recorded by all groups with respect to isokinetic contraction were not predicted. In general the literature supports the view that torque output deCreases as the velocity of contraction increases (185,219,220). However, Perrine and Edgerton (220) found their normalized torque curve to increase at slow velocity, with a relative maximum torque output occurring at approximately 96 degrees/sec. Knapnik and Ramos (274), found a high relationship between isometric tests and low

velocity isokinetic tests. In this study, the possibility that low velocity contractions allowed maximal recruitment of type I and type II fibres, while fewer type II fibres were recruited during isometric motion should not be excluded. The results may also reflect discrepencies in the test angles used to derive these data, since others have compared the force velocity relationship at constant angulation (220) or a variety of differing angles (219). Knapnik (274) suggests that fatigue may be a confounding factor in establishing the relationship between isokinetic and isometric exercise and is more likely to occur under sustained isometric conditions. Additionally, since subjects were instructed differently regarding the requirements of the contractions, factors such as neural input may have played a role in determining the torque output under the differing conditions.

TABLE 4.13: Isometric normalised torque at 30 degrees knee flexion

Means ($\bar{x}$) and standard deviation (SD) percentage values			
STAT	**NORMAL**	**RISK**	**OSTEOARTHRITIC**
$\bar{x}$	75.88	78.00	86.00
SD	31.35	26.65	48.64

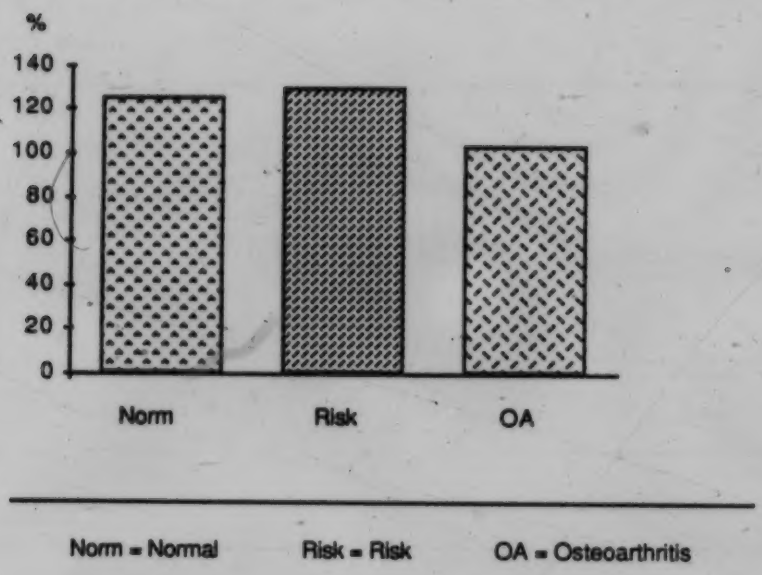

Figure 4.15. Comparison of normalised Isokinetic torque

TABLE 4.14: Isokinetic normalised torque values at 90 degrees/sec

Means ($\bar{x}$) and standard deviation (SD) percentage values of			
STAT	NORMAL	RISK	OSTEOARTHRITIC
$\bar{x}$	126.33	130.00	103.42
SD	37.26	36.93	50.95

b) Initial Torque Height-Isometric

Torque height in Nm. was measured at the end of the first recorded second of the contraction period, relative to baseline torque values at 60 and 30 degrees flexion. Raw scores were compared amongst the three groups and a normalising procedure was performed. The height comparison was employed to quantitatively determine the extent of torque production occurring in the initial period of contraction in all three groups. Normalization demonstrated the comparitive levels of tension generated amongst the three groups at 30 degrees flexion. Results of these measures are displayed in table 4.15 and figure 4.17.

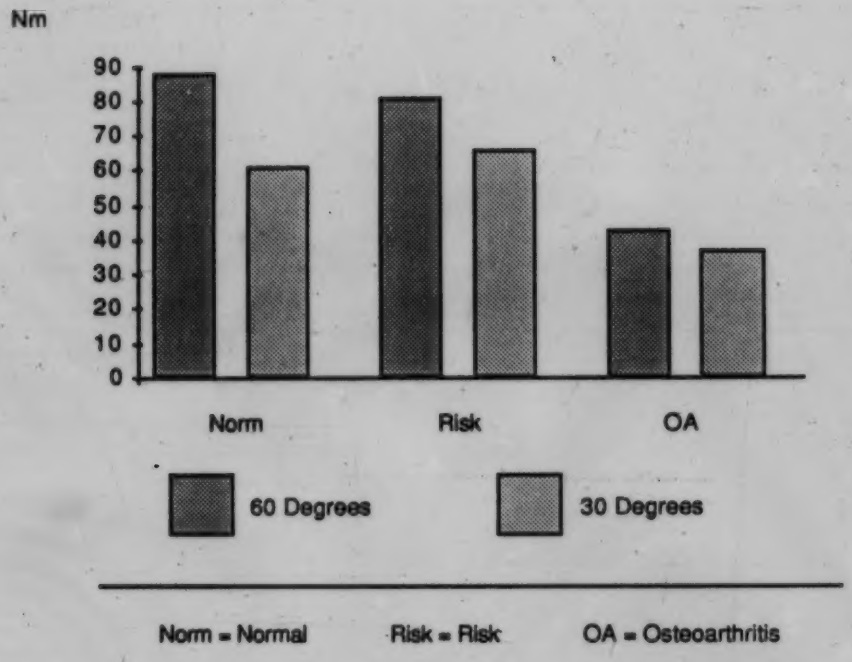

Figure 4.16. Comparison of initial Isometric torque

TABLE 4.15: Isometric torque height in 1st second

Means ($\bar{x}$) and standard deviation (SD) in Nm.
Normalised values in percent.

	STAT	NORMAL	RISK	OSTEOARTHRITIC
60 degrees	$\bar{x}$	88.38	81.48	43.10*
	SD	30.76	25.32	19.36
30 degrees**	$\bar{x}$	61.18	66.81	37.90*
	SD	24.94	26.26	18.77
Normalised	$\bar{x}$	69.00	82.00	113.00
scores	SD	8.10	9.64	10.55

*significantly different from normal and risk
**significantly different from 60 degrees

In general, arthritic subjects showed reduced raw score values of torque compared to normal and risk subjects. The relative degree of tension generation at 30 degrees was, however, considerably higher in this group. This finding confirms the trend noted in these subjects to generate relatively greater overall tension at 30 degrees flexion than either normal or risk subjects. What is also striking, is the trend of the risk subjects to generate more tension at 30 degrees flexion than normal controls, although the relative increase is still considerably lower than the trend shown by arthritic subjects. The increased

tension generated by arthritic subjects at 30 degrees, likely reflects the increasing need to stabilize the joint at this angle as previously discussed. The increased force generated by the risk group at this angle may reflect a similar need to provide improved joint stabilisation, although this idea is purely speculative.

Summary

In this study, EMG activity and time measurements relative to tension development were employed in the analysis of QF function to evaluate the extent of the muscular deficit in the osteoarthritic knee joint in this regard.

The high amplitude of peak EMG activity and the higher IEMG scores recorded by arthritic subjects were attributed to several factors. These included an increased need for muscle to provide joint stability, altered recruitment patterns and factors influencing the the normalizing process. As indicated by Brucini (36), altered patterns of EMG activity noted in persons with OA of the knee joint, both at rest and during activity, suggest heightened sensitivity of knee joint proprioceptors as a consequence of joint pathology.

The activity patterns of the RF muscle in the osteoarthritic group showed the following features. During isometric contraction RF peak activity as well as IEMG

measures were notably high. However, RF peak activity was notably decreased during isokinetic contraction, perhaps signifying loss of fast-twitch fibre content as indicated by Glasberg et al (84) and Martin et al (83).

The strong similarity between results shown by osteoarthritic subjects and reported studies on persons with chondromalacia patella are of particular interest. Since Kiss et al (252) found diffuse OA of the knee joint to begin primarily in the patellofemoral joint and evidence of improvement is noted in subjects with OA undergoing patellectomy, the underlying similarity between muscle function in OA and chondromalacia patella may provide insight into the association between these two conditions. The IEMG/force ratio values of the VM and RF muscles in the arthritic subjects were high, and may indicate their relative inefficiency during isokinetic contraction. Richards et al (276) suggest that muscle weakness will result in higher percentage utilisations i.e. increased EMG/force ratios, since it may be necessary to recruit proportionately more motor units to effect the same motor task under some conditions. In reference to the osteoarthritic knee joint, high IEMG/force values may indicate an increased need to stabilise the joint during isokinetic contraction, combined with the reduced capacity to generate extensor torque.

EMG values in all groups were generally increased

during isokinetic contraction. However speed had a different affect amongst the groups studied. While the normal group showed relatively little variation in patterns produced under isometric and slow isokinetic contractions, risk and arthritic groups showed definite differences in this regard. In particular, while activity patterns of the risk group were similar to the normal activation patterns under isometric conditions, the activity patterns of the risk group matched those of the arthritic group under isokinetic conditions. This may indicate an onset of change in the pattern of recruitment in subjects with tibial malalignment and may be of predictive value subject to further investigation.

It is also likely that the EMG patterns displayed by arthritic subjects, reflect adaptations by the central nervous system to joint pathology, as proposed by Janda (24). Here, pathological changes in the periphery (both sensory and motor) are thought to influence the central nervous system, resulting in adaptation of motor output of the muscles of the affected joint(s) in the form of altered movement patterns (24,36). The extent of the adaptive response is likely to be influenced by the degree and duration of the pathology. Afferent input from muscles, tendons, ligaments and joint receptors, as well as biomechanical demand, inflammatory processes and degree of muscle pathology may also contribute to a highly variable

pattern of EMG activity (36). While chronic partial joint fixation may reflect abnormal afferent activity from the muscle or periarticular structures as a consequence of pain (36,277), it also appears that the adaptive responses noted in this study reflect an attempt by the neuromuscular system to provide stability to the affected knee joint(s). During functional activities, it is likely that a similar demand placed upon the knee joint musculature would contribute to fatigue and low weightbearing tolerance. This effect may be considerable since the demands of weight-bearing on a flexed knee during gait may compel the patient to activate the quadriceps to a greater extent than the nonweight-bearing state (265). As confirmed by Stauffer and co-workers (31) the gait patterns of patients with diseased knee joints may reflect a compromise between the need to decrease joint pain and to increase propioceptive feedback. This increases dynamic muscular control at the expense of an apparent increase in energy expenditure.

The rate of tension development, both isometric and isokinetic showed a significant delay ($p=.05$) was incurred by the arthritic subjects. This is significant since time factors are speculated to influence the development of OA (60,61.62). Gyory and associates (278), also found timing deficits in the occurrence of floor reaction forces in subjects with diseased knees, signifying an earlier unloading of the knee during the stance phase of the

pathological lower extremity. In addition under differing mechanical conditions, the delay between the onset of electrical activity and the development of tension found is likely to be different and may contribute to excessive loading of the joint as a result (300).

Torque values calculated during the initial stages of tension development were also significantly reduced in arthritic subjects. Together with time measures noted, these findings support the view that both the rate, as well as the degree of tension development determine the susceptibility of a joint to loading (15)

CLINICAL IMPLICATIONS

Clinical observation suggests that appropiate physical intervention may provide osteoarthritic patients with significant and long-term benefits. However, there are important gaps in the knowledge on fundamental points pertinent to the application and evaluation of therapeutic intervention in OA. In particular, there is little basic knowledge concerning the function of the muscles surrounding the diseased joint. In general, past investigations of OA have focussed primarily on the tissue changes produced by the disease. Other studies have been limited to measurement of strength and endurance (30,32), muscle fibre content (83,84), needle EMG (36) and gait parameters (31,33).

The results of this study emphasise that activity patterns of the QF muscle in subjects with OA of the knee joint are significantly different from patterns of activity found in the QF muscle of normal healthy knee joints. Individual differences amongst arthritic subjects also occur and possibly reflect the presence of pain, mechanical instability and muscle weakness amongst other factors. The finding of altered movement patterns in arthritic subjects, supports the view that loss of arthrokinematic reflexs as a result of joint pathology may result in profound abnormalities of limb posture and kinetic function.

Although, the majority of studies reported in the literature describe the use of isometric exercises in the treatment of the osteoarthritic knee joint musculature, it appears therefore that patients may find the more comprehensive approaches of Gustavson (280) and Janda (24) more beneficial if movement patterns are to be normalised. Additionally, as Staudte et al (232) suggest, a treatment programme of long-lasting submaximal contractions in combination with additional maximal isometric activity which would recruit both fast as well as slow-twitch muscle fibres, may significantly enhance motor capability. Selective training of the quadriceps musculature using EMG biofeedback technology or functional electrical stimulation, may also be advantageous (83,289).

While the evaluation of torque measures provide

little about the functional status of subjects with OA of the knee and are unable to provide imformation about motor recruitment or patterns of phasic activity during movement, the evaluation of EMG activity patterns as well as torque measurements employed in this study, appeared helpful in providing imformation on muscle activity in normal and pathological joints. It is hoped that this approach will be refined in an attempt to provide physical therapists with imformation on the clinical and functional manifestations of this disease. Improved imformation in this regard, perhaps providing a better basis for the design of therapeutic intervention.

The muscular response to OA of the knee joint is complex. However, despite the complexity and variability of these responses it appears that within certain limitations, norms might be established for this group. Alternately, arthritic patients could be compared to well defined norms for an age matched population if these were available. This knowledge would help define the extent of the deficit occurring in these subjects, enhancing the specificity and outcome of the rehabilitation processes.

The potential for early prediction of OA has also been noted. The possibility for effective screening and preventative strategies for OA remains to be assessed.

V.SUMMARY AND CONCLUSIONS

The stated objectives of this study were three-fold: Firstly, to determine the EMG pattern of the quadriceps muscle of normal females and compare the pattern to females presenting with clinical signs of tibial torsion or varus malalignment. Secondly to determine whether young females with tibial torsion or varus alignment show similarities to persons who have the diagnosis of osteoarthritis of the knee joint and present with similar malalignment. Thirdly, to determine the relationship between time factors relating to tension development and the presence or abscence of pathology in these three groups.

Twenty seven female volunteers were entered into the study. Normal healthy subjects were screened during a clinical examination for their eligibility into one of two study groups. Osteoarthritic subjects were recruited by the researcher and entered into the study provided the inclusion criteria were met. One test session was held and all subjects were tested on an isokinetic dynamometer with respect to strength measures, recorded at 60 and 30 degrees flexion isometrically and at 90 degrees/sec isokinetically. In addition, EMG activity was recorded concurrently from three QF muscles, namely VM,VL and RF using surface electrodes.

The data was analysed by means of one-way and two-way ANOVA and the Newman-Keuls post-hoc test was employed

way ANOVA and the Newman-Keuls post-hoc test was employed to determine significance of the means. The level of probabilty was set at $p<.05$.

The following observations were made:

1. Subjects assigned to the risk group on the basis of joint alignment were found to differ from individuals with normal joint alignment with respect to several physical characteristics. In particular an increased height/weight ratio was noted. This finding was of interest since the arthritic subjects demonstrated extremely high values in this respect.
2. Peak EMG measures recorded isometrically showed RF activity in arthritic subjects to be significantly increased compared to normal and risk groups.
3 Total QF activity was also significantly increased during isometric contractions in this group compared to normal and risk subjects.
4. During isokinetic contraction all muscles in all groups showed heightened peak activity with the exception of RF in the arthritic group which showed a decline.
5. Total QF activity was significantly increased with respect to IEMG activity recorded in the first second of isometric contraction in arthritic subjects.
6 The RF muscle had significantly higher total IEMG values than either the total VM or VL activity at 30

degrees knee flexion.

7. At 90 degrees/sec, all muscle groups recorded significantly higher values with respect to IEMG activity.
8. Total IEMG activity of the QF muscle in the arthritic group was significantly increased with respect to normal and risk groups.
9. IEMG/force ratios were significantly higher at 30 degrees knee flexion in arthritic subjects compared to normal and risk groups.
10. In arthritic subjects, rate of tension development at 50% peak torque height was significantly different from risk and normal subjects at 60 degrees knee flexion, isometric.
11. Rate of tension development at 90 degrees/sec was significantly reduced in arthritic subjects compared to normal and risk subjects.
12. The rate of initial tension development under isometric conditions at 60 degrees knee flexion was significantly reduced in arthritic subjects compared to normal and risk subjects.
13. The rate of initial tension development under isometric conditions at 30 degrees knee flexion was considerably reduced in arthritic subjects.
14. EMD measured under isokinetic conditions was increased in arthritic subjects and decreased in risk

subjects but not statistically so.

15. There were no statistical difference in time to peak EMG activity amongst groups or muscles during isokinetic activity.
16. Normalized torque measures were lower in risk and normal subjects with reference to isometric contraction at 30 degrees knee flexion, but not statistically so.
17. Normalized torque measures for isokinetic contractions were higher than those reported in the literature for all three groups.
18. Arthritic subjects had relatively low percentage torque activity at 90 degrees /sec compared to the normal and risk groups. This was in contrast to the higher percentage noted for this group with respect to isometric exercise.
19 Torque height during the first second of contraction was significantly lower at 60 and 30 degrees isometric amongst arthritic subjects.
20. Normalized torque-height ratios were increased for risk and arthritic subjects and lower for normals.

The study of EMG patterns and time factors related to tension development was undertaken in an attempt to derive imformation on the extent of muscle pathology in female persons with OA of the knee joint. In addition, an attempt

was made to evaluate the role of tibial malalignment in the pathogenesis of the disease. No previous work employed the same premises or methods of investigation in attempting to identify causes and sequelae of OA. Within the limitations of this study, it appears that characterisation of musculoskeletal function in normals and arthritics can be aided by the use of the methodologies described. The possibilities of identifying persons at risk for OA by employing a similar approach appears to have merit based on the findings documented in this study. The imformation derived from this study indicated the need to establish a normative base from a broad spectrum of the population in order to define anthropometric and physiological variability pertinent to the understanding of OA.

This study also provided an opportunity to explore the influence of biomechanics on muscle function and to assess the association of these findings in relation to OA of the knee joint. Although the progress of muscle change with age was not evaluated and therefore the process of aging can not be excluded in the interpretation of these results, the presence of varus and/ or tibial torsion in normal healthy subjects showed similarities of EMG, time and torque measures when compared to arthritic females with similar alignment. It is concluded that the study of muscle function in normal and pathological knee joints has relevance for understanding the mechanisms of muscle

relevance for understanding the mechanisms of muscle function in the pathogenesis of OA.

Recommendations for further study.

1. The correlation between functional variables and the extent of muscle dysfunction in OA should be studied.
2. The correlation between radiographic findings and clinical measures such as EMG should be assessed.
3. Osteoarthritic subjects of both sexes should be evaluated for similarities and differences pertinent to the disease.
4. Age-matched controls should be compared to arthritic subjects to determine the extent of the similarities and differences which differentiate OA from age changes.
5. Persons with both varus and valgus as well as tibial torsion should be compared to evaluate the role of joint alignment of musculoskeletal function in gonarthrosis.
6. The evaluative protocol should be expanded to include evaluation of other knee joint musculature, as well as hip and ankle joint musculature.
7. The use of EMG as an outcome measure in clinical trials of physical therapy should be explored.
8. Time measures related to tension development such as EMD should be more thoroughly investigated in the evaluation of muscle function in normals and

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APPENDIX A

CRITERIA FOR THE CLASSIFICATION OF IDIOPATHIC OSTEOARTHRITIS OF THE KNEE JOINT*

A. CLINICAL*

This requires the following :

1. Knee pain plus at least 3 of 6 signs :

age > 50 years

morning stiffness < 30 minutes

crepitus on motion

bony tenderness

bony enlargement

no palpable warmth

95% sensitive

69% specific

B.CLINICAL AND RADIOGRAPHIC

This requires the following :

1. Knee pain plus at least 1 of 3 signs :

age >50 years

stiffness < 30 minutes

crepitus

2. Osteophytes.

91% sensitive

86% specific

3.CLINICAL AND LABORATORY

This requires the following:

1. Knee pain plus at least 5 of 9:

age > 50 years
stiffness < 30 minutes
crepitus
bony tenderness
bony enlargement
no palpable warmth
ESR < 40 mm/hour
RF <1:40
SF OA

92% sensitive
75% specific

* ESR = erythrocyte sedimentation rate (Westergren)
RF =rheumatoid factor
SF OA = synovial fluid signs of OA (clear, viscous, or white blood cell count <2000/mm^3)

** Alternative for the clinical category would be 4 of 6, which is 84% sensitive and 89% specific.

from Altman et al (44)

APPENDIX B

RADIOGRAPHIC CLASSIFICATION OF OSTEOARTHRITIS

1.RADIOGRAPHIC CLASSIFICATION OF OSTEOARTHRITIS

Grade 1: Doubtful

Small osteophytes of doubtful significance

Joint space not impaired.

GRADE 2: Minimal

Definitive osteophytic change.

Minimal joint space narrowing.

Slight Sclerosis.

GRADE 3: Moderate

Moderate dimunition of the joint space.

Some osteophytes.

Some sclerosis and cysts.

Deformity such as valgus or varus angulation.

GRADE 4: Severe

Extensive loss of joint space.

Sclerosis and cysts.

Large osteophtes.

Marked deformity.

from: Kellgren and Lawrence (48)

APPENDIX C.

QUESTIONNAIRE ON FUNCTION

PLEASE CIRCLE THE ANSWER WHICH BEST DESCRIBES YOUR PRESENT LEVEL OF FUNCTION

WALKING DISTANCE

4. Unlimited
3. 3-6 blocks
2. 1-3 blocks
1. less than 1 block

ABILITY TO GET OUT OF A CHAIR

4. Normal
3. Slight difficulty
2. Have to use arms for a boost
1. Other or unable

UP AND DOWN STAIRS

4. No problems i.e step over step without using the handrail or other support
3. No problems if the handrail or a support is used
2. One step at a time with or without support
1. Unable to go up or down at least 5 steps even with help

ASSISTIVE DEVICES

4. I do not use a cane, crutch or walker.
3. I use one cane or crutch
2. I use 2 canes or crutches
1. I use a walker.

PAIN

4. None
3. Mild pain, with fatigue
2. Moderate pain, either reducing activities or disturbing sleep
1. Constant severe pain.

APPENDIX D

KNEE ASSESSMENT FORM

KNEE ASSESSMENT FORM

SUBJECT I.D. :

AGE :

HEIGHT :

WEIGHT :

OCCUPATION :

RECREATIONAL ACTIVITIES :

DURATION of DISEASE * :

MEDICATIONS * :

	Right Knee		Left Knee	
ROM:	Flex.	Ext.	Flex.	Ext
PALPABLE T:	Yes	No	Yes	No
INSTABILITY:	Yes	NO	Yes	NO
mediolateral	----	----	----	----
anteroposterior	----	----	----	----
BONY TENDERNESS:	Yes	No	Yes	No
BONY CREPITUS:	Yes	No	Yes	No

ALIGNMENT:

	Right Knee		Left Knee	
normal	Yes	No	Yes	No
valgus	----	----	----	----
varus	----	----	----	----
Standing Flexion:	----	----	----	----
Standing Hyperextension:	----	----	----	----
Internal Tibial Torsion:	----	----	----	-----
STIFFNESS A.M.:	< 30 minutes		< 30 minutes	
	> 30 minutes		> 30 minutes	

* patients only (261)

APPENDIX E

SUBJECT CONSENT FORM

CONSENT FORM

I,_____, do hereby agree to participate as a subject in a study on muscle function in normal and arthritic knee joints to be conducted by Ray Marks under the supervision of Dr. S.J. Percy, Dr. S. Kumar and Prof. J. Semple.

The purpose of this study is to compare the activity of the thigh muscles of normal healthy women and women with osteoarthritis of the knee joint. Although this study is not being offered as a treatment for osteoarthritis, it is likely that the imformation gained from the study will be helpful to the future planning and conducting of physical treatments and disease prevention. It is also possible that results of this study will help determine the best method of treating this condition by physical therapy.

To participate in the study, I will be asked to undergo an examination of my knee joint by a rheumatologist. I will then be asked to partake in a single test session during which time a physiotherapist will test my knee muscle strength, as well as the electrical activity emitted by my knee muscles. This will involve the use of electromyography which is a commonly used diagnostic tool and is extremely safe and involves no discomfort.

I will be familiarised with all test equipment before the test and will be allowed to practice the different test movements as well. These include 2 types of exercises which will be performed while I am seated and secured in an exercise chair. During these exercises small discs will be placed on my thigh muscle in order to record the electrical activity of this muscle.

During the practice and test I will be asked to perform several contractions of my knee joint muscles lasting 3 seconds or less and I will be given adequate time to rest at all times.

As with all exercise, it is possible that I might feel some soreness or stiffness the next day. In addition there is a possibilty the exercise may increase the discomfort in my knee temporarily. However, since I am only being asked to contract my muscle for less than one minute (much less than in normal activity), the probability of incurring pain or having it last longer than 1-3 days is very slight.

The imformation obtained in this study bearing my name will be seen only by the principal investigators. Any imformation that is shown to others, published or presented at conferences will not refer to me by name, but only by number and only when this is necessary. All imformation will become the property of the Department of Physical therapy, University of Alberta.

I understand that the investigator will freely and willingly answer any questions I may have concerning the study and my participation in it.

I may also decline the study or withdraw from the study at any time without any effect on my medical treatment or any other penalty.

In the event that questions arise concerning the study, I will be free to contact Mrs. Ray Marks, at 432-2068 (work) or 439-2297 (home).

With my signature below, I certify that I understand what will be required of me in the study, and I also acknowledge receipt of a copy of this consent form.

---------- ----------

Subject's signature Investigators signature

---------- ----------

Witnesses signature Date

(261)

APPENDIX F.

CYBEX CALIBRATION

CYBEX CALIBRATION

Recorder Divisions	Lever Arm Inches	Weight Pounds	Torque Input Foot Pounds	Graph Scale
360	30	70.0	180	5 major
180	31	32.5	90	5 major
30	33	5.0	20	20 minor

Lever Arm is equivelant to the distance from the centre of the Cybex input shaft to the centre of the T-tube (lever arm length).

Calibration Procedure

1. Turn on power and allow for five minutes warm -up.
2. Select appropiate scale to be calibrated (0-30, 0-180 or 0-360).
3. With speed selector on at 30 degrees per second and the recorder ON, but no torque applied to the lever arm:
   a) Select #3 position on Damping control.
   b) Select slow chart speed 5mm/s).
   c) Align stylus with baseline of chart grid paper using "Zero Adjustment Button".
   d) Check to see baseline does not shift when range scale is changed from 180 to 360. Baseline shift of

this nature can be corrected by adjusting with small screwdriver, the potentiometer on the top right side of the recorder (marked zero null).

4. Attach balance weighed disc weights to the T-tube at the lever arm length indicated for the scale to be calibrated as per above calibration.

5. Dynamic calibration is performed manually lifting the weighted to the vertical position and allow gravity to swing it down until the weights contact the floor. As the weighted arm passes the horizontal, the graph recording will show this value as a maximum point on the curve. If this point is above or below the correct torque value, adjust the recorder and make it read the correct value by turning the appropiate potentiometer (30, 180 or 360) through the holes on the top right side of the recorder (marked accordingly). Turning the potentiometer clockwise will increase the reading and counter clockwise will decrease it (241).

APPENDIX G

SPECIFICATIONS OF THE LEAF ELECTRONICS SYSTEM VII ELECTROMYOGRAPH

SPECIFICATIONS OF THE EMG SIGNAL CONDITIONER

1. Battery operated and therefore isolated from AC source
2. Gain 1:20 ratio set at varying levels i.e.200,500,1,000.
3. Impedence-input = 1 mega ohm: output =100 ohm.
4. Frequency response ; 10 Hz - 10 KHz.
5. Common Mode Rejection Ratio (CMMR) : 90 DB
6. Filter : internal - 60 -120 Hz. reject
   external - wide band pass.
7. Time constant- variable.

APPENDIX H

CONFIRMATION LETTER

TO: Volunteers who have agreed to participate in the study to examine the electromyographic activity of the knee joint muscles during extension movements.

Dear :

Thank you for being so willing to act as a subject in this study. It has been possible to arrange your test appointment as follows:-

Your appointment will be on:

_____ at _____

I would be grateful if you could let me know in advance if for any reason you are unable to attend your appointment. You may get in touch with me by leaving a message in the graduate student mail box, 210 Corbett Hall, Department of Physical Therapy or by telephoning 432-2068 or 439-2297 and leaving a message.

You may feel free to invite a colleague or a friend to accompany you to the test session if you wish, since they may be glad of an opportunity to see what is being done.

Please bring a pair of shorts or very loose pair of slacks so that the knee joint and the muscles above can be suitably exposed. You may wear running shoes or walking shoes for the test.

I look forward to seeing you

Yours Sincerely, Ray Marks. Adapted from (299)

APPENDIX I

EMG CALIBRATION

CALIBRATION PROCEDURE

1. Charge battery overnight,and allow unit to rest unused for one full day following charging before beginning calibration.
2. Turn all channel gain preset potentiometers to minimum i.e full counter-clockwise as viewed from the knob side of the potentiometers located nearest rear panel.
3. Set monitor to the channel being calibrated, set meter to sample and hold position.
4. Set front panel gain to zero on all channels.
5. Set rear panel integrator calibration setscrews at maximum clockwise.
6. Set oscilloscope on 5mv/division vertical sensitivy and zero with shortened leads.
7. Connect D.C. scope to integrator output on channel being calibrated.
8. Set artifact suppression off.
9. Set volume at zero, but turned "ON".
10. Set time constants at 100 msec.
11. Set rectifier gain control (1 Meg linear pot) at maximum i.e. full clockwise.
12. Adjust integrator offset control until there is minimum D.C. offset at integrator output.
13. Connect scope to rectifier output.

14. Adjust rectifier offset for minimm D.C> at rectifier output.
15. Return scope to integrator output, re-zero integrator offset.
16. Return scope to rectifier output, re-zero rectifier offset.
17. Repeat last two steps until less than 1mV of offset appears at either output.
18. Repeat the entire procedure for remaining three channels, being careful not to disturb previously-adjusted settings.
20. Re-check all outputs for drift after 1/2 hour of being "ON" has elapsed.

BATTERY CHECK

1. When the power is switched on, the internal power pack should be tested. The meter should read in the region of "6" to indicate adequate power. If either positive or negative readings are too low, the battery requires recharging.

APPENDIX J

RAW DATA

RAW DATA:PEAK ISOMETRIC EMG

SUBJECT	VM	VL	RF
1.	111	111	130
2	97	68	94
3.	127	116	71
4.	102	95	113
5.	125	140	113
6.	100	120	100
7.	92	108	112
8.	107	96	103
9.	137	126	121
10.	85	100	125
11.	146	136	142
12.	183	191	157
13.	118	100	110
14.	94	100	100
15.	75	100	133
16.	106	125	100
17.	141	143	122
18.	77	84	103
19.	125	100	200
20.	100	75	95
21.	88	114	125
22.	80	66	200
23.	206	180	185
24.	240	185	200
25	160	185	150

RAW DATA: ISOKINETIC PEAK EMG

SUBJECTS	VM	VL	RF
1.	140	155	170
2.	77	44	100
3.	151	127	121
4.	107	111	113
5.	137	162	131
6.	116	160	145
7.	120	141	161
8.	123	103	109
9.	162	147	121
10.	85	71	100
11.	164	126	157
12.	191	216	185
13.	200	144	120
14.	211	275	225
15.	125	116	166
16.	116	175	100
17.	291	231	178
18.	96	109	138
19.	50	33	100
20.	83	75	108
21.	133	157	200
22.	320	233	150
23.	160	150	142
24.	280	257	228
25.	106	157	175

RAW DATA: ISOMETRIC IEMG

SUBJECT	VM	VL	RF
1.	75	89	100
2.	104	79	80
3.	82	85	115
4.	122	219	214
5.	85	74	110
6.	125	92	113
7.	117	163	106
8.	166	161	177
9.	95	77	146
10.	97	80	116
11.	72	93	111
12.	175	178	148
13.	100	146	146
14.	110	125	475
15.	100	195	108
16.	166	75	134
17.	46	80	66
18.	80	72	100
19.	500	392	231
20.	103	175	140
21.	85	66	555
22.	169	126	228
23.	151	148	173
24.	157	166	100
25.	61	50	26

RAW DATA:ISOKINETIC IEMG

SUBJECTS	VM	VL	RF
1.	115	136	168
2.	166	155	130
3.	148	147	200
4.	163	288	253
5.	125	137	126
6.	153	139	232
7.	135	238	153
8.	285	234	244
9.	225	184	442
10.	175	151	167
11.	161	175	134
12.	224	189	229
13.	364	538	396
14.	262	250	565
15.	96	236	78
16.	357	177	192
17.	166	195	193
18.	110	193	181
19.	611	421	318
20.	159	171	186
21.	200	500	666
22.	179	176	328
23.	233	229	360
24.	42	16	100
25.	53	27	100

RAW DATA:ISOMETRIC IEMG/FORCE RATIO

SUBJECTS	VM	VL	RF
1.	75	89	100
2.	88	67	66
3.	81	79	114
4.	144	250	251
5.	164	145	211
6.	240	173	213
7.	91	126	80
8.	247	235	264
9.	90	73	135
10.	114	160	157
11.	156	100	121
12.	195	198	167
13.	111	165	165
14.	87	100	416
15.	115	220	123
16.	195	76	135
17.	130	130	92
18.	141	69	171
19.	377	292	363
20.	216	369	300
21.	142	110	555
22.	169	126	228
23.	105	104	121
24	458	496	296
25.	61	50	26

RAW DATA: ISOKINETIC IEMG/FORCE RATIO

SUBJECTS	VM	VL	RF
1.	79	100	121
2.	124	115	96
3.	109	105	142
4.	254	400	400
5.	113	125	113
6.	176	152	268
7.	100	173	114
8.	221	179	188
9.	94	118	182
10.	227	220	250
11.	136	145	113
12.	278	236	288
13.	207	337	248
14.	256	302	433
15.	96	135	160
16.	717	305	338
17.	130	158	153
18.	100	94	157
19.	1300	892	672
20.	52	56	64
21.	426	187	364
22.	100	98	156
23.	100	32	227
24.	53	27	100

RAW DATA: ISOMETRIC TRTD AT 50% MVC (mm/sec) (Conv factor 3.88Nm/mm)

SUBJECTS	NORMAL		RISK		OA	
	60	30	60	30	60	30
1.	62	40	116	75	60	40
2.	70	50	60	40	30	15
3.	37	80	65	62	1	1
4.	65	75	31	20	11	4
5.	75	53	60	80	23	23
6.	63	90	22	47	8	13
7.	48	80	30	50	7	4
8.	18	40	47	17	3	10
9.	20	27	35	23	30	17

RAW DATA: ISOKINETIC TRTD (mm/sec) (Conversion 3.88Nm/sec)

SUBJECTS	NORMAL	RISK	OA
1.	70	23	24
2.	85	93	26
3.	85	93	1
4.	68	86	-
5.	110	92	50
6.	106	47	46
7.	87	133	20
8.	100	83	63
9.	75	90	

RAW DATA: ISOMETRIC TRTD (MM/SEC) (Conversion 3.88Nm/mm)

SUBJECTS	NORMAL		RISK		OA	
	60	30	60	30	60	30
1.	140	24	27	18	30	8
2.	23	30	40	20	40	37
3.	26	43	28	24	1	1
4.	47	65	28	23	12	5
5.	67	43	75	60	30	23
6.	63	50	25	35	9	16
7.	23	40	70	43	7	5
8.	18	16	45	15	4	5
9.	10	14	19	35	16	11

RAW DATA:INITIAL TORQUE -ISOMETRIC (Nm)

	NORMAL		RISK		OA	
SUBJECT	60	30	60	30	60	30
1.	20	10	35	30	12	18
2.	13	19	15	11	22	9
3.	21	19	26	23	10	6
4.	39	30	17	11	13	8
5.	28	15	17	23	13	17
6.	28	13	16	14	7	9
7.	23	14	22	18	7	4
8.	16	8	17	11	5	6
9.	17	14	24	14	12	11

RAW DATA: ISOKINETIC EMD (msec)

SUBJECTS

	VM	VL	RF
1.	10	1	10
2.	5	5	5
3.	10	10	10
4.	5	5	5
5.	20	20	0
6.	18	18	0
7.	30	30	5
8.	40	40	1
9.	10	10	10
11.	1	12	1
12.	5	0	10
13.	10	10	-
14.	15	15	50
15.	10	10	-
16.	40	40	5
17.	8	8	1
18.	10	10	-
19.	10	10	-
20.	1	1	1
21.	10	10	10
22.	20	20	20
23.	30	30	30
24.	30	30	30

RAW DATA:TIME TO PEAK EMG (secs)

	GROUP	MUSCLE	TIME
1.	1	VM	1.1
2.	1	VM	.7
3.	1	VM	1.1
4.	1	VM	.7
5.	1	VM	.8
6.	1	VM	.6
7.	1	VM	.75
8.	1	VM	1.2
9.	1	VM	.8
10.	1	VL	.7
11.	1	VL	.8
12.	1	VL	1.0
13.	1	VL	.8
14.	1	VL	.9
15.	1	VL	.7
16.	1	VL	.7
17.	1	VL	.8
18.	1	VL	.4
19.	1	RF	.45
20.	1	RF	.7
21.	1	RF	1.0
22.	1	RF	.45
23.	1	RF	.6
24.	1	RF	.7
25.	1	RF	.75
26.	1	RF	.8
27.	1	RF	1.0
28.	2	VM	1.0
29.	2	VM	.5
30.	2	VM	.5
31.	2	VM	.9
32.	2	VM	.8
33.	2	VM	.9
34.	2	VM	.7
35.	2	VM	.6
36.	2	VM	.9
37.	2	VL	.7
38.	2	VL	.9
39.	2	VL	.5
40.	2	VL	.85
41.	2	VL	.75
42.	2	VL	.85
43.	2	VL	.75
44.	2	VL	.55
45.	2	VL	.90
46.	2	RF	.5
47.	2	RF	.85
48.	2	RF	.6
49.	2	RF	.65
50.	2	RF	.65

51.	2	RF	.85
52.	2	RF	.6
53.	2	RF	.7
54.	2	RF	.9
55.	3	VM	.6
56.	3	VM	.3
57.	3	VM	1.1
58.	3	VM	.85
59.	3	VM	1.6
60.	3	VM	.9
61.	3	VM	.7
62.	3	VL	.2
63.	3	VL	.2
64.	3	VL	1.1
65.	3	VL	.85
66.	3	VL	1.0
67.	3	VL	.7
68.	3	VL	.6
69.	3	RF	.4
70.	3	RF	.3
71.	3	RF	1.0
72.	3	RF	.85
73.	3	RF	.9
74.	3	RF	.3
75.	3	RF	.7

APPENDIX K

ANOVA SUMMARY TABLES

PEAK EMG:ISOMETRIC

Source	SS	df	MS	F	p
group	18117.678	2	9058.839	7.39	.0016
muscle	1753.129	2	876.565	.715	
group muscle	3936.772	4	948.193	.803	
Error	80874.430	66	1225.370		

PEAK EMG:ISOKINETIC

Source	SS	df	MS	F	p
group	15283.962	2	7641.981	2.313	.1052
muscle	229.060	2	114.530	.035	
group muscle	975.815	4	243.954	.074	
error	218085.016	66	3304.318		

IEMG:ISOMETRIC

SOURCE	SS	df	MS	F	p
group	59145.184	2	29572.592	3.388	.03
muscle	18979.221	2	9489.610	1.087	.3440
group muscle	3933.691	4	983.423	.113	
Error	576150.31	66	8729.550		

IEMG:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	35799.062	2	17899.531	1.032	.36
muscle	34996.840	2	17498.420	1.009	.37
group muscle	11847.424	4	2961.856	0.171	
Error	1144226.500	66	17336.766		

IEMG/FORCE RATIO:ISOMETRIC

SOURCE	SS	df	MS	F	p
group	133607.328	2	66803.664	6.308	.00
muscle	21365.986	2	10682.993	1.009	.37
group muscle	2136.522	4	534.130	0.050	
Error	699013.120	66	10591.108		

IEMG/FORCE:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	145394.031	2	72697.016	1.728	.18
muscle	21344.223	2	10672.111	0.254	
group muscle	46884.535	4	11721.134	0.279	
Error	2650008.500	63	42063.629		

TRTD:ISOMETRIC (50%mvc)

SOURCE	SS	df	MS	F	p
group	15471.448	2	7735.724	16.470	.00
angle	8.165	1	8.165	0.017	
group angle	602.771	2	301.386	0.642	
Error	22545.109	48	469.690		
Total	38627.492	53			

TRTD:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	17047.2852	2	8523.6426	22.067	.0
Error	8883.8750	23	386.2554		
Total	25931.1602	25			

INITIAL TRTD:ISOMETRIC

SOURCE	SS	df	MS	F	p
group	7038.259	2	3519.130	7.419	.00
angle	840.168	1	840.168	1.771	.186
group angle	98.112	2	49.056	0.103	
Error	22767.334	48	474.319		
Total	30743.875	53			

NORMALISED TORQUE:ISOMETRIC

SOURCE	SS	df	MS	F	p
group	512.0859	2	256.0430	0.189	
Error	32484.8945	24	1353.5372		
Total	32996.9800	26			

NORMALISED TORQUE:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	3407.778	2	1703.8889	0.997	
Error	37601.7030	22	1709.1683		
Total	41009.4800	24			

TORQUE HEIGHT:ISOMETRIC

SOURCE	SS	df	MS	F	p
group	919.001	2	459.500	11.351	.00
angle	220.020	1	220.020	5.435	.02
group angle	72.702	2	36.351	.898	
Error	1943.111	48	40.481		
Total	3154.835	53			

EMD:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	653.166	2	326.583	2.184	.11
muscle	395.964	2	197.982	1.324	.27
group muscle	283.509	4	70.877	0.474	
Error	8972.339	60	149.540		

TTP EMG VM:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	.0629	2	.0315	.437	
Error	1.5847	22	.0720		
Total	1.6476	24			

TTP EMG VL:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	.0430	2	.0215	.406	
Error	1.1658	22	.0530		
Total	1.2088	24			

TTP EMG RF:ISOKINETIC

SOURCE	SS	df	MS	F	p
group	0.0313	2	.0157	.283	
Error	1.2186	22	.0554		
Total	1.2499	24			

PEAK EMG:ISOMETRIC

SOURCE	SS	df	MS	F	p
group	18117.9043	2	9058.9521	7.60	.0014
Error	85804.5620	72	1191.7300		
Total	103922.4690	74			

PEAK EMG RF:ISOMETRIC

SOURCE	SS	df	MS	F	p
group	15266.6602	2	7663.3301	10.25	.001
Error	16371.9893	22	744.1813		
Total	31638.6484	24			

APPENDIX L

RELIABILITY COEFFICIENTS

RELIABILITY COEFFICIENTS FOR PEAK EMG RECORDINGS

Peak EMG values in mm. were calculated for three muscle groups and two contraction speeds. Four readings were compared over two trials with the electrodes being removed and replaced for the second trial. Measures were recorded at a single session with a 20 minute intertrial period.

Isometric 30 degrees

	Trial I	Trial II	
VM	8	10	
	15	17.5	
	15	15	
	7.5	10	(r =.96)
VL	10	10	
	12.5	12	
	17.5	15	
	11	12.5	(r =.89)
RF	8.5	8.5	
	15	10	
	15	15	
	10	7.5	(r =.90)

ISOKINETIC

	Trial 1	Trial 2	
VM	13	19	
	13	11	
	15	18	
	15	20	(r = .96)
VL	20	20	
	25	24	
	17.5	15	
	22	25	(r = .89)
RF	17.5	17.5	
	15	10	
	15	15	
	20	15	(r =.90)